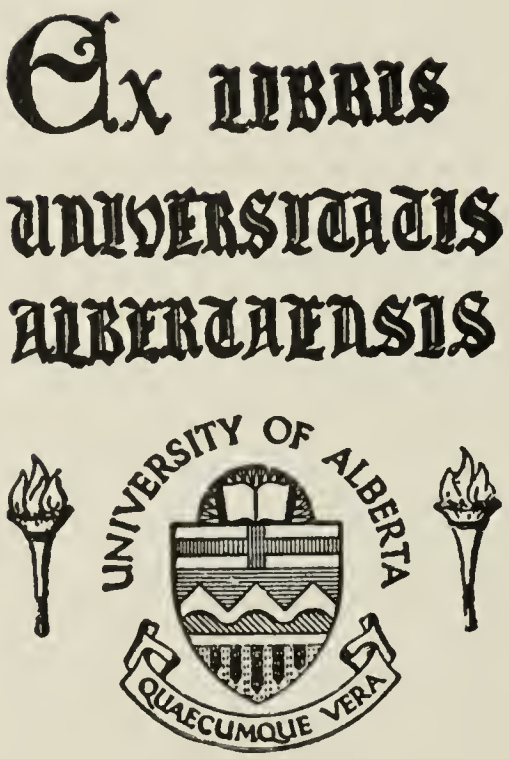

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THE EFFECT OF ELECTRO-CONVULSIVE THERAPY ON REMINISCENCE AND KINESTHETIC FIGURAL AFTEREFFECTS

by

David Lyle Rennie

A THESIS

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "The Effect of Electro-convulsive Therapy on Reminiscence and Kinesthetic Figural Aftereffects", submitted by David Lyle Rennie in partial fulfilment of the requirements for the degree of Master of Arts.

Abstract

Two groups of fifteen psychiatric patients were tested on two occasions to determine the neural effect of electro-convulsive therapy (ECT). One group received ECT between the testing occasions, the other group served as a control. An attempt was made to discriminate between a cortical facilitation hypothesis and a brain damage-inhibition hypothesis of the neural effect of ECT. Two measures of cortical inhibition (Ward-Hovland reminiscence and kinesthetic figural aftereffects) were used to make this discrimination. An hypothesis was advanced that a state of cortical facilitation resulting from ECT would decrease the tendency to develop central inhibitory state and would be manifested by a decrease in the magnitude of reminiscence and of kinesthetic figural aftereffects. It was conversely hypothesized that a temporary brain damage, probably producing an inhibitory state, would increase the level of central inhibition and would be manifested by increased reminiscence and kinesthetic figural aftereffects. Neither hypothesis was supported by the results of the study. However, the results suggested that ECT impairs acquired habits through an interference with memory mechanisms but without interfering with the capacity to acquire subsequent new learning.

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David Lyle Rennie

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CHAPTER I

INTRODUCTION

Theories of the Effectiveness of Electro-convulsive Therapy

Electro-convulsive therapy (ECT) was introduced into modern psychiatry by Cerlitti and Bini in 1938. Since that time it has proved to be an effective treatment for certain psychiatric disorders, particularly depressive states. The effectiveness of the treatment is empirically determined, however, and up to the present time its *modus operandi* remains speculative (Campbell, 1962). A number of theories have been advanced to explain its operation, some of which are psychodynamic, others being neuro-physiological. One psychodynamic theory cited by Campbell (1962) suggests that ECT acts as a punishment which satisfies the patient's need for atonement. A second psychodynamic theory maintains that ECT acts by increasing ego repression of disturbing psychic material (Alexander, 1953).

The neurophysiological theories can be divided into those which maintain that ECT acts by producing an excitatory state in the cortex and in this way facilitating the subject's behavior, and into those which maintain that ECT interferes with the memory mechanisms of recently learned maladjustive habits and thereby allowing older non-maladjustive habits to be re-established.

A representative of the cortical excitation group of neuro-logical theories is that put forward by Gellhorn *et al* (e.g., Gellhorn and Kessler, 1941; Gellhorn, Kessler and Minatoya, 1942; Kessler and Gellhorn, 1943). These authors suggest that ECT

stimulates the sympathetic centers in the hypothalamus, producing activation of the sympathetic division of the autonomic nervous system (ANS) and also an upward discharge from the hypothalamus to the cortex, causing a state of cortical facilitation. Also, Sargant (1957), basing himself on Pavlovian theory, claims that ECT produces an extreme cortical excitatory state, leading to an ultraparadoxical phase of behavior with a reversal of conditioned reflexes.

The neurophysiological theories which stress an interference with newly acquired habits (Duncan, 1948) hold the view that ECT causes a temporary brain damage, which view has been supported by physiological studies of animals subjected to electro-shock convulsions (Morrison, Weeks and Cobb, 1923; Heilbrunn, 1943). This brain damage may affect memory mechanisms either by an increase in the overall state of cortical inhibition held by several authors to be concomitant with brain damage (Kohler and Fishback, 1950; Klein and Krech, 1952; Eysenck, 1955), or by an interference with the consolidation of memory traces (Thompson and Dean, 1955).

According to Campbell (1962), none of the above, or any other view, has been adequately demonstrated. The aim of the present study was to clarify the issue between two of the neurophysiological theories, namely, the facilitation as opposed to the brain damage-inhibition hypothesis.

The Facilitation Hypothesis. The facilitation hypothesis was introduced by Gellhorn and his associates in a series of papers which reported the effects of ECT upon the ANS and later, upon Pavlovian conditioning. Using blood sugar levels and other homeostatic mechanisms as criteria, Gellhorn et al claim that metrazol as well as electro-convulsive shock stimulates both the sympathetic and parasympathetic divisions of the ANS, with the sympathetic action being predominant. This suggestion was supported by analyses of the effects of ECT when either division of the ANS was altered.

The major mediators for sympathetic activity are the hormones emanating from the adrenal medulla, whereas the vagus nerves constitute the important pathways for parasympathetic activity. It was found that increased blood sugar levels, indicants of sympathetic arousal which usually followed convulsions, were prevented by extirpation of the adrenal medulla. After elimination of the adrenal medulla, convulsive procedures lowered the blood sugar but this effect was absent when the vagus nerves were cut.

Blood pressure changes, as well, were considered to support the interpretation that ECT causes predominant sympathetic activity. An initial fall, of vagal origin, was followed by a prolonged rise due to increased sympathetic discharges (Piette, 1951). The shortening of blood clotting time after electro-shock (Walther, 1949) suggested, in the light of Cannon's studies (1941), secretion of adrenalin and chemical analyses of the blood showed that the adrenalin content of the blood was increased (Kinzius and Hann, 1951).

Gellhorn et al postulate that the sympathetic arousal is initiated by activation of the sympathetic centers in the hypothalamus. It is further reasoned by Gellhorn that, since there are pathways from the hypothalamus to the cortex through thalamo-cortical projections, a stimulation of the hypothalamic sympathetic centers would cause a spread of excitation, or "upward discharge", into the cortex (Gellhorn, 1953) and would produce facilitation of cortical activity. This theory of cortical facilitation was supported by Pavlovian conditioning studies. It was found that when a conditioned response had been extinguished through a series of non-reinforced trials, the application of electro-shock re-established the conditioned response (Gellhorn, Kessler and Minatoya, 1942; Gellhorn and Kessler, 1943; Gellhorn, 1945). In terms of Pavlovian theory as represented by Gellhorn et al, this discovery was an example of disinhibition which was produced by the dominance of excitatory processes over the inhibitory processes which had caused the extinction of the response.

In the light of more recent discoveries, it may be postulated that the dominance of the excitatory processes was produced in its turn through the facilitation of cortical activity by the ascending reticular activating system (ARAS). It has been demonstrated that there are complex interactions between the ARAS and the rest of the central nervous system. One of these interactions is a "neural feed-back" mechanism between the ARAS and the autonomic nervous system. As summarized by Gooch (1963), fluctuations in the level

of activation as indicated by electro-encephalogram recordings have been observed to be associated with fluctuation in general autonomic tone (Bonvallet, Dell and Heibel, 1954). Activation tends to be accompanied by a release of adrenal hormones, which in turn have an action on the adrenalin sensitive portions of the midbrain activating system tending to potentiate the activation of the cortex.

Thus the facilitation hypothesis maintains that the stimulation of the sympathetic centers of the hypothalamus, which can be inferred from an increased activity of the peripheral sympathetic nervous system, produces, through an upward discharge via the ARAS, cortical arousal and facilitation, manifesting themselves as Pavlovian disinhibition causing a reappearance of extinguished conditioned responses.

However, while there is weighty evidence that ECT causes a re-establishment of the balance between the sympathetic and para-sympathetic division of the ANS, the behavioral evidence concerning the facilitation of the cortical activity is not conclusive. Further conditioning studies (Gellhorn, 1945) revealed that if two conditioned responses were extinguished, it was the most thoroughly learned response which was disinhibited, regardless of the temporal order of conditioning. These results can be better understood in the light of three subsequent studies by other investigators. Duncan (1948) found that if an animal was conditioned to replace one habit with another, electro-convulsive shock (ECS) caused a reversal to the

earlier learned habit. His results are explained by the suggestion that electro-shock removes recently acquired habits, allowing older ones to reappear. In a related study, Thompson and Dean (1955) found that a discrimination habit was increasingly impaired as the time interval separating the learning of the habit and the administration of ECS diminished. They conclude that ECS impairs the consolidation of the memory traces associated with learned responses. On the other hand, a study by Braun and Patton (1950) demonstrated that Duncan's explanation was true only when the earlier learned habit was less difficult to solve than the later habit. If the reverse were the case, the animal would, following ECS, display the habit which was most recently learned prior to the administration of electro-shock.

The selection of the least difficult of a repertoire of habits following ECS as outlined by Braun and Patton is similar to the concrete behavior of subjects suffering from brain damage (Goldstein and Scheerer, 1941). The impairment of the memory traces associated with learned responses also points in the direction of neurological damage, consequently there is an alternate explanation, unrelated to disinhibition, for the results of the conditioning studies of Gellhorn et al.

The concept of ARAS arousal resulting in cortical facilitation is related to Hull's theory of 'drive' (Hull, 1943) and Malmo's concept of 'arousal' (Malmo, 1957), hence behavior subsequent to ECT predicted by the psychological theories would tend to validate the facilitation hypothesis. Hull's theory states that the excitatory

reaction potential of a learned response is monotonically related to the level of drive. Malmo, on the other hand, found that performance is a function of arousal but that this function is not monotonic but inverted U-shaped. Thus, if ECT causes a state of over-arousal, task performance, instead of showing a generalized improvement, may become disrupted. However, unless there is an extreme arousal, Gellhorn's theory of upward discharge from the hypothalamic sympathetic centers would predict a state of cortical facilitation resulting in a generalized improvement of behavioral functioning.

The evidence is conflicting in this regard. It has, of course, been shown that depressed patients are less depressed after ECT. The remission of depression could be interpreted as improved functioning. But other behavioral indices have demonstrated functional impairment. There is a failure to respond to any kind of stimuli immediately following the grand mal convulsion, with a period of gradual return of psychological functions during which signs typical of "organic" impairment can be evoked (Stainbrook, 1944; Flescher, 1942). The retrograde amnesia apparently disappears within a few hours (Campbell, 1962) but, when reinforced by motivational factors, some amnesias can persist for as long as three months (Hanis, 1950). Animal studies have shown that ECS causes a reduction in overall activity (Mirsky and Rosvold, 1953; Stern, 1956) and impairments in the abilities to solve complex tasks (Russell, 1949). Human learning studies

have revealed the ECT causes decrements in performance (Callagan, 1952; Campbell, 1957).

Physiological Evidence of Brain Damage after ECT. Many of the functional deficits cited above are attributed to the effects of a temporary brain damage resulting from ECT. There is physiological evidence that such damage does occur and is due to haemorrhaging in the meninges and within the brain (Morrison, Weeks and Cobb, 1923), brought about by changes in circulatory pressure during the convulsion (Heilbrunn, 1943). However, such evidence is not conclusive. For instance, Seikert, Williams and Windle (1950), having found no differences in the post-shock cranial autopsies of six monkies and one control, suggested that the results of the previous investigators were artefacts of poor surgical techniques.

Summary of the Apparent Effects of ECT. At this point a brief summary of the foregoing discussion is in order. There is evidence that ECT causes sympathetic arousal in the ANS; disturbances in the evocation of conditioned responses; remission from depressive states but certain functional deficits. The apparent sympathetic arousal may be due to the action of ECT on the sympathetic centers in the hypothalamus and the facilitatory centers in the ARAS, which would affect the central nervous system (CNS) as well as the ANS. On the other hand, ECT seems to cause a temporary brain damage, possibly due to cerebral haemorrhaging, which may account for deficits of functioning following the treatment. It is not known which, if either theory of the neural effects of ECT is tenable.

The Role of Neural Inhibition Theory. The theoretical issue could be clarified if, in some way, measures were given which would discriminate between the facilitatory and decremental effects of ECT at the behavioral level. Such measurements are difficult to obtain because the behavioral correlates of neural processes have not been specifically defined. In the present study, nevertheless, it was considered that a general framework was provided by the neural inhibition theories, such as neural satiation (Kohler and Wallach, 1944) and reactive inhibition (Hull, 1943). Each of these theories proposes that prolonged sensory stimulation increases the cortical resistance to response evocation which, according to some writers (e.g., Klein and Krech, 1952; Eysenck, 1955), becomes more pronounced in the case of brain damage.

Satiation theory was developed to explain perceptual distortions following prolonged exposure to a figure or object, known as figural aftereffects. An example of aftereffects in kinesthetic perception occurs when a subject first grasps a rectangular wooden block between thumb and forefinger for a few seconds. This stimulation is followed by a period of prolonged rubbing, once again between thumb and forefinger, of a wider block. When the first block is subsequently grasped, it will seem to be narrower than it did the first time. Figural aftereffects have been demonstrated not only in kinesthesis but in vision (Gibson, 1933) as well.

Kohler and Wallach maintain that the contours of the inspection figure are insomorphically projected on the cortex, regarded by these

authors as a volume conductor. The difference in the electrolyte concentrations between the cortical area of the figure projection and its background cause a difference in depolarization of the cortical cell membranes with a resultant flow of the so-called figure electric currents. It is theorized that prolonged stimulation of the same cortical region results in a state of relative depolarization, analagous to the condition of electrotonus in physics, described as satiation. A satiated area in the cortex is an area of increased resistance. Figure currents, like all direct currents, are assumed to flow in the lines of least resistance. Consequently, when the contours of a second inspection stimulus are projected on a previously stimulated and therefore satiated, area of the cortex, the new figure currents will be displaced away from the area of satiation to a region of lower resistance. It is this displacement which is subjectively experienced as the figural aftereffect.

Reactive inhibition, symbolized I_R, was developed by Hull (1943) to explain performance decrements during prolonged practice at learning tasks. This construct was based upon a number of earlier observations and hypotheses, chief of which were the Mowrer-Miller hypothesis relating to work, Pavlov's internal inhibition, the Ward-Hovland phenomenon of reminiscence[1] and the

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[1]The term 'reminiscence' was first used in experimental psychology when Ballard (1913) found that children who learned poetry recalled it better after a delay of two or three days

(Footnote continued next page)

performance difference resulting from massed (continuous) as opposed to distributed (spaced) practice at motor learning tasks. It was likened to a negative drive state in that it reduces reaction potential and accordingly depresses performance (Duncan, 1956). It was assumed to occur with every repetition of a response, whether it was reinforced or not, and to spontaneously dissipate with the passage of time. It is most commonly measured by Ward-Hovland reminiscence, which is an improvement in the initial level of performance following rest from massed practice at a learning task (e.g., Ammons, 1947; Kimble, 1952). I_R was originally interpreted to be a peripheral phenomenon, similar to "fatigue" but more recent studies have demonstrated bilateral transfer of reminiscence in motor learning, which suggests that the process has a central locus (Grice and Reynolds, 1952; and Irion and Gustafson, 1952).

(Footnote 1 continued)

than was possible immediately after learning. This type of reminiscence came to be known as the Ballard-Williams Phenomenon, which must be distinguished from the Ward-Hovland Phenomenon. As described by Osgood (1953), the Ward-Hovland Phenomenon refers to a temporary improvement in performance, without practice, appearing over short intervals of between 2 and 5 minutes. The Ballard-Williams Phenomenon is similar in that it refers to improvement in performance without practice, but is different in that the temporal interval over which it appears is much longer, favorable delays being as great as 2 or more days.

A number of studies have suggested a relationship between brain injury and the development of neural satiation. Bender and Teuber (1947, 1948) reported that in the visual field of patients in whom parts of the occipital brain had been injured, the perceived objects were displaced from their normal positions. Referring to this study, Kohler and Fishback (1950) suggest that the displacement had the same direction as it would have had in figural aftereffects, therefore the injured part of the cortex could be regarded as a highly satiated area. The inference is made that, quite apart from any strictly local disturbance of function, the injured tissue of such patients produces a highly polarizable area.

Klein and Krech (1952) reported that brain injured subjects showed greater figural aftereffects than did those whose brains were not injured. They used the kinesthetic test as a measure of figural aftereffects. They draw a parallel between brain damage and satiation as produced by continuous stimulation. A suggestion is made by them that whereas satiation results in temporary and localized reduction in conductivity, brain injury produces a permanent and generalized state of lowered conductivity (basal inhibition). They maintain that the properties of figural aftereffects measure this conductivity, so that the rate of appearance, extent and persistence of figural aftereffects would be greater in brain damaged cases than in normals (Meyer, 1962).

Eysenck (1955a, 1955b) identifies satiation with Hull's concept of reactive inhibition.[2] According to Eysenck's theory, in the sphere of personality, subjects in whom strong reactive inhibition is generated quickly and dissipated slowly develop hysterical types of disorders in neurotic breakdown; conversely, individuals in whom weak reactive inhibition develops slowly and dissipates quickly are predisposed to develop dysthymic disorders. Clinical evidence indicates that the pattern of symptoms of the hysteric is similar to the pattern of symptoms of the brain injured person. Petrie (1952) provided some evidence that brain injury (surgical interference by frontal leucotomy) produces a marked shift in the direction of greater extraversion as measured by objective tests. This finding, Eysenck argues, is in agreement with the hypothesis that brain injury produces an increase in the speed and strength of reactive inhibition as a concomitant of the measured increase in extraversion (Meyer, 1962). Eysenck also cites the results of the Klein and Krech (1952) study as supporting his hypothesis.

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[2]This identification of the two hypothetical processes is supported by Duncan (1956). He reports that reactive inhibition and neural satiation would seem to be describing the same neural process because they both (1) are consequences of afferent stimulation; (2) have cortical loci; (3)distort behavior away from some criterion or standard; (4) accumulate to measurable amounts, given task differences, within five to ten seconds; (5) develop rather quickly to a kind of maximum; and (6) begin to dissipate very rapidly after cessation of stimulation.

It must be emphasized that the question of the identity of the neural processes responsible for the phenomena of reactive inhibition and satiation; the question of the nature of these processes; and the question of their exact relation to the effects of brain injury are far from being finally settled. Osgood and Heyer (1951) attack Kohler and Wallach for postulating and entirely novel set of non-neuronic electrical forces as the basis of satiation theory. They maintain that Kohler and Wallach's conception of direct currents within a field instead of a passage of electrical impulses along nerve fibres is at odds with accepted neurophysiological knowledge. Osgood and Heyer propose an alternate interpretation of satiation, based upon the Marshall-Talbot statistical theory of the distribution of neuronal excitations in the cortex.

Howarth (1957), using two tests of figure-reversal, was unable to duplicate a number of Kohler's findings concerning the development, direction and permance of satiation.

Broadbent (1958) takes issue with reactive inhibition and submits that many extinction phenomena, usually explained by I_R, can equally well be accounted for by the organism's capacity to "filter" competing responses. Eysenck (1963), on the other hand, maintains that the "filter" hypothesis, while plausible when perceptual data alone are considered, breaks down when attempts are made to explain reminiscence and other similar phenomena.

The Present Study

The present study was designed to differentiate between the neural facilitation and brain damage-inhibition theories of the effectiveness of ECT by using a measure of reactive inhibition and a measure of neural satiation. In keeping with the postulate Kohler and Fishback (1950); Klein and Krech (1952); and Eysenck (1955), it was assumed that an increase in cortical inhibition is a concomitant of brain trauma. Also, following the postulate of Eysenck (1955) and Duncan (1956) it was assumed that reactive inhibition and neural satiation are constructs which describe the same central inhibitory process. It was further assumed that the hypothetical inhibitory process resulting from brain trauma in consequence of ECT is either similar to the inhibitory process described by reactive inhibition and neural satiation, or influences the tendency to develop this process.

In reference to the facilitation hypothesis, it was assumed that an increase in central excitation as a result of reticular arousal would decrease the tendency to develop the central inhibitory process described by reactive inhibition and neural satiation.

Reminiscence (Ward-Hovland Phenomenon) following rest from massed practice at the pursuit rotor learning task was used as a measure of reactive inhibition, whereas a test for kinesthetic figural aftereffects provided a measure of neural satiation. Based on the foregoing assumptions, an hypothesis was advanced

that if ECT causes a state of cortical facilitation, the putative central inhibitory process responsible for reminiscence and kinesthetic figural aftereffects would be reduced causing a decrease in the latter phenomena in subjects who have undergone ECT. Conversely, if a state of cortical deficiency, probably inhibitory in nature, resulting from a temporary brain damage is produced by ECT, it was predicted that both reminiscence and kinesthetic figural aftereffects would increase in these subjects.

CHAPTER II

METHOD

Subjects

Experimental group: Fifteen patients from the psychiatric wards of a university hospital were included in the experimental group. These patients were tested before and after they received ECT. The group consisted of eleven women and four men, ranging in age from 23 to 58 years, with an average age of 45.4 years. The diagnoses of the patients are subsumed under the general categories of endogenous depression (N = 6); involutional melancholia (N = 2); neurotic depressive reaction (N = 4); schizophrenia (N = 2).[1] The lengths of stay in the hospital prior to the time of testing ranged from one to 31 days, with an average length of stay of 8.3 days.

Control group: Fifteen patients from the same wards of the hospital who did not receive ECT served as a control group. This group consisted of thirteen women and two men, ranging in age from 23 to 49 years, with an average age of 33.1 years. The diagnoses of these patients are described by the general categories of endogenous depression (N = 3); involutional melancholia (N = 1); neurotic depressive reaction (N = 5); neurotic anxiety reaction (N = 1); homosexuality (N = 1) and schizophrenia (N = 3).

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[1]The reader is referred to Appendix A for a complete list of psychiatric diagnoses given to the patients

The lengths of stay in hospital before testing for this group ranged from one to ten days with an average duration of 5.9 days.

In neither group were patients with mental deficiency, known neuro-organic disease or severe psychotic states included.

Table 1 summarizes the mean ages and lengths of hospitalization before testing of the two groups. The table shows that there were no significant differences in lengths of stay but that the two groups were statistically different in age ($\underline{t} = 3.40$; $p < .01$). This age discrepancy resulted from sampling difficulties was due to several factors, chief of which was an inability to control the treatments given to subjects between the time when they first underwent testing and the time, approximately two weeks later, when they were retested. This difficulty necessitated an exclusion from the sample of some of the control subjects, originally matched with the experimental subjects as to age, diagnosis, length of hospitalization and the time interval between the two testing occasions.

The inability to control the treatments given to the patients created the additional difficulty that most of the subjects were medicated at the time of each testing occasion. The danger of confounding the effects of ECT with the effects of drugs was considered a serious difficulty in the study. However, it was thought that the drug effects would be randomized throughout the two groups and further, that each subject receiving the same

TABLE 1

MEAN AGE AND LENGTH OF STAY
BEFORE TESTING FOR ALL SUBJECTS

(N = 15 in each group)

Group	Age in Years		Days Hospitalization Prior Occasion 1	
	$\bar{X}$	S.D.	$\bar{X}$	S.D.
Experimental	44.8	10.37	7.3	7.96
Control	33.1	9.55	5.9	3.39

Note: $\underline{t}$ for the age difference between groups = 3.40; ($p < .01$).

$\underline{t}$ for the hospitalization difference = 0.63 (NS).

medication before and after treatment would serve as his own control. However, by the time testing was completed, it was apparent that there had been some variability in the drugs given to the members of the experimental group when compared to those given to the members of the control group. Therefore, analyses were conducted to compare the types and dosages of drugs given to the two groups of subjects.

The drugs administered to the subjects which had action upon the CNS or the ANS were either tranquilizers, antidepressants or sedatives. The sedatives were usually given at bedtime and exerted an effect for approximately eight hours: the testing of the subjects for the present study was conducted in the afternoon or early evening, prior to night-time sedation, thus only those patients receiving day-time sedation were considered to be influenced by the action of this type of drug at the time of testing.

In comparing the drugs administered to the two groups, it was necessary to take into account not only the type of drug given but also the dosage strength. For the sake of simplicity, the dosage of a drug administered to a given patient was compared to the 'usual dosage' range as described by the manufacturer of the drug in Vademecum 1964. A drug dosage which corresponded to the recommended 'usual' dosage range was given an arbitrary weighting of I. Dosages which exceeded the usual range were classified as 'large' and were given an arbitrary weighting of II (patients who were given extremely large dosages were not

included in the study). The use of arbitrary weightings in this way, while providing no absolute measures of dosages, did provide indices whereby the relative dosages given to each group could be compared.

The comparisons of the types and dosages of drugs given to the two groups of subjects were carried out in the following manner. The drugs and their dosages prescribed to a given patient during the four day period before each occasion were selected as the criteria of his medication at the time of testing[2]. The three categories of drugs were kept discrete. For each category the two groups were contrasted in terms of the number of subjects who (1) did not receive the drug; (2) were given usual dosages; and finally, (3) were given heavy dosages. Separate analyses were conducted for the three types of drugs for each occasion using the Chi Square test for two independent samples.

A summary of the Chi Square analyses of the occurrence of each type of drug within the two groups at the time of each occasion is shown in Table 2. It can be seen that the only significant difference between the groups concerned the greater use of antidepressants by the experimental subjects before the first occasion ($\chi^2 = 7.49$; $p < .05$). The implications of this difference are discussed in a later section.

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[3]The specific drugs given to each patient at the time of each occasion are listed in Appendix A.

TABLE 2

SUMMARY OF χ^2 TESTS OF DIFFERENCES BETWEEN GROUPS REGARDING DRUGS RECEIVED AT TIME OF EACH OCCASION[3]

Drug	χ^2	
	Occasion 1	Occasion 2
Antidepressants	7.49*	3.66
Tranquilizers	5.62	2.40
Sedatives	2.62	3.33

[3]Complete tables are shown in Appendix B,

*$p < .05$

Apparatus.

A. Measure of Reminiscence. Measures of reminiscence were obtained from a Lafayette pursuit rotor with a ten inch turntable rotated by an electric motor at either 30, 45 or 60 RPM. A smooth metal disc, three-fourths of an inch in diameter, even with the surface of the turntable, was located one inch from its edge. The disc was electrically charged by a 25 volt direct current, transformed from 120 volt A.C. by either of two Hunter timers. A stylus, consisting of a hook-ended metal rod hinged to a wooden handle, was connected by wire to an electrical outlet on the rotor apparatus. Contact between the stylus tip and the metal disc closed the circuit and was recorded by one of the Hunter timers to within one thousandth of a second. A Lafayette repeat cycle timer (Model No. 2) alternated the current flow from one Hunter timer to the other every ten seconds and thus divided a massed practice period into trials of that duration.

The performance task with this apparatus required the subject to keep the stylus tip on the disc as much as possible while the turntable rotated. A performance score during a given ten second trial period consisted of the percentage time the stylus remained on target.

The majority of pursuit rotor studies to date have used a turntable speed of 60 RPM. However, in a pilot project preliminary to the present study, it was found that 60 RPM created a task which was too difficult for most hospital patients, therefore a rotation rate of 45 RPM was adopted.

B. Measure of Kinesthetic Figural Aftereffects. The kinesthetic figural aftereffect (KAE) equipment consisted of two rectangular wooden blocks, twenty inches in length and two inches deep, surfaced with non-gloss paint and supported by inverted-T stands four inches in height. The Test block, to which the subject was exposed before and after stimulation with an Inspection block, was two inches in width. The Inspection block was one inch wider than the Test block. The subject was blindfolded to eliminate visual cues concerning the width of the Test block. He judged the width of the Test block by moving his hand up or down a wooden wedge, 41 inches in length and tapering in width from four inches at its wide end to one-eighth of an inch at the opposite end. A cloth tape was glued to the top of the wedge to indicate to the experimenter the width of the wedge as it tapered from its wide to its narrow end. The tape indicated one-sixteenth of an inch changes in the width of the wedge. Hence, once the subject stopped exploring the wedge and came to the place which he judged to be the same width as that of the Test block, (the point of subjective equality, or p.s.e.) through the use of the tape the experimenter recorded the subject's p.s.e. to within one-sixteenth of an inch. For example, instead of judging the Test block to be two inches in width, the subject may have judged it to be 1 7/16 in., and so on.

A given block and the wedge were placed upon tables 26 inches in height, which, in combination with the heights of the inverted-T

stands upon which the blocks or wedge were supported, raised the stimulus surfaces of the objects to the level of the subject's hands when his arms rested by his sides. The tables were covered with felt so that the test objects could be soundlessly moved or replaced.

Procedure

Testing was conducted in a 10 by 14 foot room furnished with two desks, two chairs and a filing cabinet. The pursuit rotor apparatus was situated on a desk; the KAE equipment was located at the other end of the room and was hidden from view by a draped sheet.

Each subject was tested on two occasions, before and after treatment. The average delay between occasions was 13.8 days for the experimental group and 18.0 days for the control group. For pursuit rotor learning during each occasion there were two periods of five minutes massed practice separated by a ten minute rest. The test for KAEs followed a ten minute rest after the last period of massed practice during each occasion.

Once in the testing room the subject was shown the pursuit rotor equipment with the following instruction. "Here we have a revolving turntable with a metal disc on it." (The turntable was manually spun and the disc was pointed out.) "The disc is sensitive to the touch of this stylus, so that whenever they make contact, it will be recorded on those timing machines over there. Do you understand? Good. Now what I want you to do is

to hold this stylus in your hand like this. When the turntable spins around (the rotor was switched on), try to keep the tip of the stylus on the disc like this (demonstration). Make sure that you move your whole arm around, like this." (At this point the subject usually gripped the wooden handle of the stylus too close to the metal hinge, endangering a closing of the electrical circuit because the subject served as a ground, so the caution had to be made:) "Make sure that you hold the handle far enough back so that you don't touch any metal part." (Also, most subjects found it easier to keep the stylus on the target if they exerted pressure on the stylus by tilting it at right angles to the handle, locking the hinge, thus the additional caution had to be made:) "Make sure that you keep the stylus handle horizontal, or flat, with the turntable, like this, so that it is free to move up and down. The slightest touch will be recorded so that you won't have to worry about pressure. Are you ready? Good. (Examiner sat down in front of the timers, which were placed oblique to the subject's line of vision so that he could not see his score.) Once you start, don't stop until I give you the word. Alright, go ahead."

Once started, some subjects had difficulty hitting the target. Complaints were usually answered with "It takes a while. Keep going. You'll get on to it." Sometimes, however, a subject threatened to quit the task before the time was up. On such occasions, if encouragement such as "There's just a little while longer. Keep going." failed, the examiner resorted to

instructions such as "Try to move your whole arm a little more. That's it." Subjects repeatedly had to be admonished to keep the stylus horizontal with the turntable.

When the first period of massed practice was finished, the subject filled out a questionnaire (the Maudsley Personality Inventory) during the ensuing ten minute rest interval between practice periods. Some subjects completed the questionnaire in five minutes or less, others failed to finish it during the first rest interval and continued on with it during the rest interval between the last period of massed practice and the KAE test. Those subjects who completed the questionnaire were given magazines to read for the remainder of the rest interval.

The KAE test followed the last rest interval. The subject was blindfolded, the draped sheet was drawn away from the equipment and the subject was made to stand between the two tables which supported either the Test or Inspection blocks on his left and the wedge on his right. For the standardization trial, he grasped the Test block between his left thumb and forefinger and, moving his right hand up or down the wedge, located the place where the two widths seemed to be the same (i.e., the point of subjective equality or p.s.e.). He let go of the wedge and stood at rest for one minute. Then, keeping his right hand free, he lightly rubbed the Inspection block between his left thumb and forefinger at the rate of approximately one rub per second for forty seconds. The Inspection block was immediately replaced with the Test block and he once again located the p.s.e.

Since the Inspection block was wider than the Test block, after stimulation with the Inspection block, the Test block should have seemed narrower to the subject than it did before stimulation with the Inspection block. The difference between judged width of the Test block after stimulation with the Inspection block and the initial judged width of the Test block was the KAE score. As outlined in an above section, the KAE scores were measured to within 1/16 of an inch.

There were three potential sources of error which had to be controlled in the KAE procedure. The first source of error was the direction of approach to the p.s.e. on the wedge. It is sometimes found that if a subject approaches the p.s.e. from the wide end of the wedge, he will tend to "stop more toward the wide end". In the present study, the Test block was narrower than the Inspection block so that one would expect a reduced p.s.e. (figural aftereffect) following stimulation with the Inspection block. If a subject stopped closer to the wide end because he approached the p.s.e. from that direction, the after-effect would have been neutralized. Conversely, an approach from the narrow end may have prompted him to stop closer to that end which would have created a spurious aftereffect. Other researchers (Klein and Krech, 1952; Eysenck, 1955) have attempted to control this problem by giving the subject some practice trials before the test trial is given. But this procedure introduces a second source of error, that of order effect, i.e., the subject's

experience during the previous trial changes his perception of the situation in the subsequent one. There is a danger that order effects, if extreme, will distort the phenomenon being studied.

In the present procedure, potential contamination by the direction of approach was controlled by changing the direction of approach for each successive subject in either group. In other words, the first experimental subject approached the p.s.e. from the wide end of the wedge, the second from the narrow end, etc. The danger of order effects was eliminated by giving a single trial to the subject. This latter procedure weakened the reliability of the measurement. However this drawback was compensated by giving precise instructions to the subject (see below). Further, the subject was encouraged to touch the narrow and wide ends of the wedge prior to approaching the p.s.e. so that he would know where the extreme limits were.

The third potential source of error was that of arm position effects. There was a possibility that, during the test trial after stimulation with the Inspection block, the subject would judge the p.s.e. as being the place where his arm was before, i.e., during the standardization trial. This potential source of error was first counteracted by encouraging the subject to respond to how the block felt between his thumb and forefinger. Secondly, he was made to stand so that his arms, hanging loosely, were opposite the center of the stimulating block on his left. The point of actual equality (p.a.e.) on the wedge was then moved three inches either forward or backward in relation to the center of the

stimulating block on the left during the standardization trial and in the opposite direction for the test trial. The direction of the movement of the wedge, too, varied from one subject to another.

In order to control the last two sources of error, subjects were allocated at random to each of the four following procedures: (1) Approach from the narrow end of the wedge, wedge in the backward position; (2) approach from the narrow end of the wedge, wedge in the forward position; (3) approach from the wide end of the wedge, in the backward position; and (4) approach from the wide end of the wedge, in the forward position. For the sake of clarity, it must be emphasized that for a given subject, the direction of approach during the standardization and test trials remained the same but the position of the wedge changed.

The manner in which a given subject was instructed in the test will now be described. Once between the two tables supporting the equipment, the position of his stance was adjusted until his left hand, hanging free, was parallel the center of the Test block on his left. He was given the following instruction: "I would like you to stay relaxed and to keep your hands by your sides for a moment. You are standing between two tables with wooden blocks on them. The block on your left is rectangular shaped, the one on your right is wedge shaped, or tapered, in other words it is wide at one end and narrow at the other. Can you imagine a block like that? Good. Now what I will want you to do in a

minute is this. I will want you to grasp the rectangular block on your left between your thumb and forefinger and then move your right hand either down or up the wedge until you come to the spot that seems to be the same width, or thickness, as the rectangular block. Do you understand? Alright. Now, for the moment, keep your left hand by your side. The wedge is narrow on this end (the examiner grasped the subject's wrist and directed his hand) and wide on this end. Now I want you to grasp the other block between your left hand, like this, and then starting from this end of the wedge, try to find the spot that is the same width. Go ahead. Let me know when you are finished."

When the subject had located the p.s.e. the examiner said "Good. Now let go of both of the blocks, rest your arms by your sides for a minute." During the one minute rest, the examiner replaced the Test block with the Inspection block and noiselessly shifted the position of the wedge. When the minute ended, he said, "Alright... Now this time I want you to use just your left hand. Keep your right arm by your side. I want you to rub this block between your thumb and forefinger at about this rate of speed (the examiner took hold of the subject's wrist and moved his hand back and forth along the block at a pace of about one rub a second) until I tell you to stop. Are you ready? Go ahead."

When the rubbing period was over, the examiner immediately replaced the Inspection block with the Test block. As this was being done, the subject was told: "Now, I am going to have you

judge the width of this block, on the wedge, like you did the first time. Now since that time, I have moved the position of the wedge but that shouldn't affect you, simply judge the width of the wedge as it feels between your thumb and finger. Alright? Go ahead. Start from this end again (the examiner guided the subject's hand to the appropriate starting end of the wedge). When this second p.s.e. was located, the testing was completed.

CHAPTER III

RESULTS

The pursuit rotor performance curves for the two groups of subjects during the pretreatment and post-treatment testing occasions are shown in Figure 1. Reminiscence for each group during either occasion was measured by comparing the first twenty second trial of the post-rest period of massed practice with the last twenty second trial of the pre-rest period.[1]

A summary of the analysis of variance for the group performancce scores immediately before and after rest during both occasions is shown in Table 3. It can be seen that there was a highly significant main effect for periods (C) indicating significant differences between the last pre-rest and first post-rest trials for each group during both occasions ($F = 26.17$; $p < .01$). Both before and after treatment, the performance following the rest from massed practice was significantly higher than that before the rest period, hence reminiscence was characteristic of the two groups before and after treatment.

The average reminiscence scores for the control group were 14.7 and 7.07 for the pretreatment and post-treatment occasions respectively, as compared to 7.47 and 9.53 for the experimental

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[1]The ten second trials which made up the raw data for pursuit rotor performance during a massed practice period were grouped into twenty second trials to reduce variability.

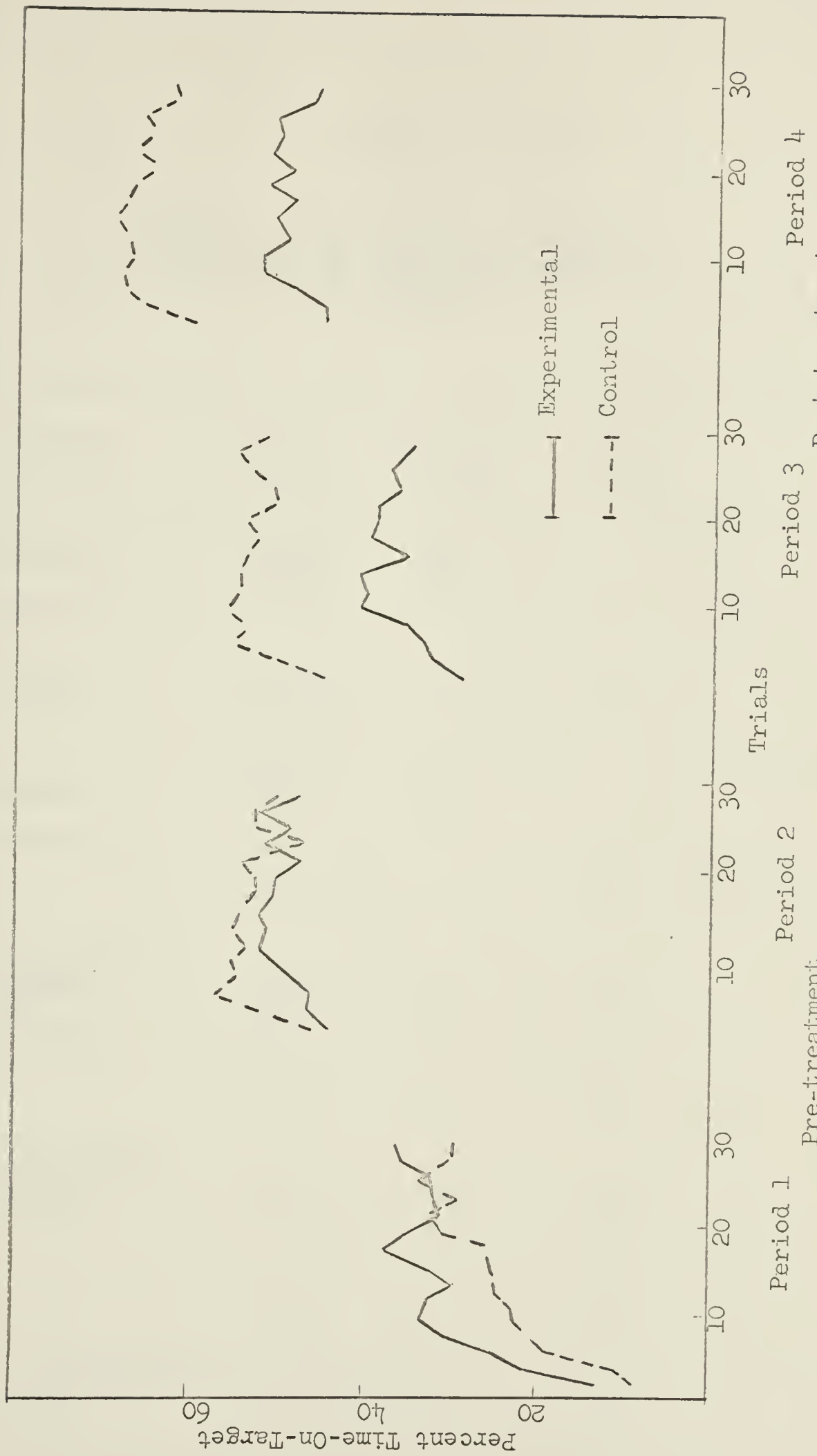

Fig. 1. Pursuit rotor performances of the pre-treatment and post-treatment occasions.

TABLE 3

SUMMARY OF THE ANALYSIS OF VARIANCE OF PRE-REST AND POST-REST TRIALS OF EACH OCCASION

Source of Variation	Sum of Squares	Degrees of Freedom	Mean Square	F
Between Ss	25432	29		
A (Groups)	1321	1	1321	1.54
Ss within groups	24021	28	858	
Within Ss	20398	90		
B (Occasions)	1937	1	1937	6.42*
AB	2465	1	2465	8.16**
B X (Ss within groups)	8422	28	301	
C (Periods)	2843	1	2843	26.17**
AC	45	1	45	0.41
C X (Ss within groups)	5042	28	108	
BC	60	1	60	1.20
ABC	184	1	184	3.70
B X C X (Ss within groups)	400	28	50	

* Significant at the .05 level

** Significant at the .01 level

group during the two occasions. It is clear that the control subjects exhibited more reminiscence than did the members of the experimental group during the first occasion but slightly less reminiscence during the second occasion. However, these differences in the amounts of reminiscence between the two groups are not statistically distinguishable as is apparent from the lack of a significant groups by periods[2] interaction effect in the analysis of variance.

Referring again to Table 3, it is evident from the nonsignificant occasions by periods interaction that for either the experimental or control group there was no significant difference between the amount of reminiscence developed during the post-treatment occasion and the amount of reminiscence developed before treatment. Similarly, the lack of a significant groups by occasions by periods interaction shows that there was no statistical distinction between the difference in reminiscence before and after treatment for the experimental group and the difference in reminiscence between occasions for the control group.

These results which demonstrate a lack of chance in reminiscence between occasions for the experimental group failed to confirm the general hypothesis put forward in this study, that ECT would affect the cortical process responsible for reminiscence.

While there were no significant differences either within or between the groups regarding the amount of reminiscence developed

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[2]Geometric representations of the A X C and B X C interactions are shown in Appendix C.

within the two testing occasions, there were significant differences in the general levels of performance for the two groups between occa ions. More specifically, Table 3 shows that of the main effects there was a significant occasion effect ($F = 6.42$; $p < .05$). Further, there was a significant groups by occasions interaction ($F = 8.16$; $p < .01$) which suggests that there was a significant difference between the two groups in terms of the general level of performance before and after treatment. The performance of the two groups was not different during both periods of massed practice for the pretreatment occasion.[3] However, the performance of the experimental subjects after ECT was lower than that of the control subjects for both the pre-rest and post-rest periods of practice.

In order to establish that kinesthetic figural aftereffects occurred in the sample of subjects tested, differences between the scores before and after satiation were ranked for both groups and the Wilcoxen matched-pairs signed ranks test was carried out.[4]

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[3]The results of four separate analyses of variance and trend analyses which were carried out to compare the performance and learning characteristics of both groups during each period of massed practice are presented following the KAE results.

[4]Nonparametric statistics were used because it was not possible to balance the conditions of trials mentioned in the section on the procedure (p. 30) and it was considered that ordinal scores were safer to use than those implying interval measures.

The distributions of kinesthetic figural aftereffect scores before and after treatment and the tests of significance are shown in Table 4. The results indicate that significant KAE scores occurred for the experimental group during the first occasion ($T = 4$; $p < .005$). The aftereffect phenomenon was also generally prevalent for the control group during this occasion but the scores were short of significance ($T = 17.5$; $p < .1$). Significant KAEs were characteristic both the experimental group ($T = 2.5$; $p < .005$) and the control group ($T = 9.5$; $p < .01$) during the post-treatment testing occasion.

After the presence of KAE had been established for each occasion, a comparison was made between the amount of KAE developed by the experimental group and the amount developed by the control group. The Mann-Whitney U test was used to make the two comparisons. Secondly, for each group a comparison was made between the amount of KAE developed during the first and second occasions. The Wilcoxen matched-pairs signed ranks test was used for these comparisons. Also, the Mann-Whitney U test was carried out to test for the difference between the groups in the amount of change in KAE between the two occasions. The results are summarized in Table 5. It can be seen that there were no significant differences between the two groups in terms of the sizes of the KAEs developed during either the first or second occasion. Nor were there significant changes in aftereffects for either group between the first and second occasions. This last result, in the case of the experimental group, parallels the absence of change in reminiscence and

TABLE 4

DISTRIBUTION OF KAE SCORES RESULTING FROM THE DIFFERENCE BETWEEN PRE-INSPECTION AND POST-INSPECTION SUBJECTIVE WIDTHS OF TEST BLOCK

Subject Group	Score[1] -6	-5	-4	-3	-2	-1	0	1	2	3	4	5	6	7	8	T[a] for Difference
Occasion 1																
ECT				1				3	2	2	2	4			1	4**
Control	1			1	1	1	3		2	1	1			2	2	1	17.5
Occasion 2																
ECT					1		3		2	2	3	1	1	1		2.5**
Control				1	1	1	3		2	1	3			1	1	1	9.5*

*$p < .01$
**$p < .005$

[1]The scores are scaled from -0.375 inches to 0.500 inches with 0.0625 inch intervals in the scale.

[a]Wilcoxen matched-pairs signed ranks test

TABLE 5

COMPARISONS OF GROUP DIFFERENCES IN MAGNITUDES OF KINESTHETIC FIGURAL AFTEREFFECTS (KAEs)

(N = 15 in each group)

KAE Comparison	Statistic	Level of Significance
Comparison between ECT and Control group		
Occasion 1	$\underline{U}^a = 93$	N.S.
Occasion 2	$\underline{U} = 102.5$	N.S.
Comparison between Occasions 1 and 2		
ECT group	$T^b = 55.5$	N.S.
Control group	$T = 42.0$	N.S.
Comparison of groups in change of KAE	$\underline{U} = 108$	N.S.

[a]Mann-Whitney $\underline{U}$ test.

[b]Wilcoxen matched-pairs signed ranks test.

accordingly further suggests that ECT has no effect on the development of cortical inhibition, provided that the measures of it used in this study were valid.

The foregoing concludes the presentation of results concerning reminiscence and kinesthetic figural aftereffects. These experiments tested the facilitation and inhibition hypotheses of the effects of ECT put forward in the introduction to this thesis. Neither of the above hypotheses was supported by the results of the experiments. In examining the learning curves of the pursuit rotor task, however, it became apparent that ECT affected performance on this task, which was not specifically hypothesized before the experiments were conducted.

Closer attention will now be paid to the performance and learning characteristics of the two groups during each period of massed practice before and after treatment. Table 6 includes the F tests resulting from the analyses of (1) the differences between the performances of the two groups during each period of massed practice; (2) the significance of the linear, quadratic and cubic components of the learning trends within each period[5] and finally, (3) the significance of the interactions between the two groups in terms of the linear, quadratic and

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[5]The linear component describes the slope of a learning curve; the quadratic component its curvilinearity and the cubic component the amount of "S"-shape within the curvilinearity.

TABLE 6

TABLE OF F TESTS FOR PURSUIT ROTOR PERFORMANCE AND LEARNING DIFFERENCES BETWEEN THE GROUPS FOR EACH PERIOD OF MASSED PRACTICE

(N = 15 in each group)

Type of Analysis	Massed Practice Period			
	1	2	3	4
(1) Analysis of Variance				
A (Groups)	0.96	0.25	4.36*	8.05**
B (Trials)	19.76**	0.88	4.09**	4.86**
AB	1.80*	0.43	0.49	0.9
(2) Significance of Trend Components				
Linear	83.13**	0.95	2.40	0.04
Quadratic	13.10**	8.51**	11.77**	14.07**
Cubic	3.65	3.42	5.49*	2.76
(3) Difference Between Groups re Components				
Linear	13.83**	0.59	0.52	0.17
Quadratic	1.98	1.33	2.65	2.15
Cubic	0.92	0.30	0.71	0.95

* Significant at the .05 level

** Significant at the .01 level

cubic components of the trends. It is evident from the table that during the pre-rest period of practice during the first occasion there was no significant difference between the groups regarding the levels of performance. There was a significant trials[6] effect during this period ($F = 19.76$; $p < .01$) which according to the trend analysis, was due to significant linear ($F = 83.13$; $p < .01$) and quadratic ($F = 13.10$; $p < .01$) components of the curves. The significant linear component can be assumed to reflect the marked learning to be expected during the first period of practice at a task, whereas the quadratic component indicates a significant reduction in the slope of the curves during the latter trials of the period. Of greater interest for this period of practice is the significant groups by trials interaction ($F = 1.80$; $p < .05$) due to a difference in the linear components of the curves ($F = 13.83$; $p < .01$) which suggests that the two groups of subjects learned at different rates. Visual inspection of the curves shows that the experimental subjects learned more rapidly than did the control subjects.

If the assumptions made in this study[7] about the meanings of the linear and quadratic components are correct, there were no significant differences between the groups in learning during the

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[6]In order to carry out the trend analysis the 30 ten second trials which made up the raw data for each period of massed practice were grouped into five one-minute trials.

[7]See footnote page 41.

post-rest period of practice before treatment. Further, there was no difference between the groups in overall performance for this period of practice. After treatment, however, the performance of the experimental group was significantly lower than that of the control group for both the pre-rest ($F = 4.36$; $p < .05$) and post-rest ($F = 8.05$; $p < .01$) periods of practice. This result confirms the presence of such a decrement indicated earlier by the analysis for reminiscence. This decrement is further illustrated when for each group the initial trial of the first period practice post-treatment is compared to the final trial of the last period pre-treatment. There is no statistical distinction between these two trials for the control group ($\underline{t} = 0.91$) whereas it is highly significant for the experimental group ($\underline{t} = 4.77$; $p < .001$). Also, the mean difference between the above two trials for the experimental group was significantly greater than the mean difference of the control group ($\underline{t} = 1.99$; $p < .05$).

While there was a performance decrement after ECT, there was no apparent difference between the groups in the shapes of the learning curves for each period of practice during the second occasion, as is indicated by the nonsignificant F ratios for the significance of the differences between groups regarding the trend components. This similarity in the shapes of the learning curves is one indication that there was no difference in learning between the two groups during the second occasion. A second

indication of the lack of difference between the groups regarding the capacity to learn during the second occasion was obtained when the maximum gain in performance for the members of each group was compared. The criterion of maximum gain for each subject was the difference between the first 20 sec. trial of Period 3 and highest 20 sec. trial of either Period 3 or 4. As it turned out, the average maximum gain (learning score) of the post-ECT group was 35.1, which was slightly greater than the control group score of 31.9 (t = 0.74; N.S.).

Upon completion of the above analyses concerning the performance and learning differences of the two groups were completed, one further analysis was made. There was a possibility that the performance decrement after ECT on the part of the experimental group was due not to the effect of ECT but to the confounding effect of antidepressant drugs, since there was a significantly greater incidence of this type of drug in this group (see p. 21). The effect of antidepressant drugs on performance during the pre-treatment occasion was determined by combining the two groups and then separating those subjects who received antidepressants (N = 11) from those who did not (N = 19). For the first occasion, the average performance scores of the antidepressant group were 36.4 and 60.1 for the pre-rest and post-rest practice periods, respectively, in contrast to 20.3 and 48.9 for the non-antidepressant group. This performance superiority on the part of the antidepressant group was significant for the pre-rest period (t = 3.39; $p < .01$) and

approached significance for the post-rest period ($\underline{t}$ = 1.46; $p < .2$). It is thus evident that the antidepressants had a facilitatory, rather than a decremental, effect on performance.

CHAPTER IV

DISCUSSION

The results of the present study have failed to confirm the hypothesis that ECT affects the central inhibitory process responsible for reminiscence and kinesthetic figural aftereffects. The fifteen subjects of the experimental group demonstrated no significant change in either reminiscence or the KAE scores following ECT when compared to the scores prior to treatment. Furthermore, there was no difference between the experimental and control groups in the amount of reminiscence and KAEs developed during either occasion. Consequently, provided the assumption that reminiscence and figural aftereffects are measures of an inhibitory, neurological process is correct,[1] neither the facilitation nor the brain damage-inhibition hypothesis of the effects of ECT on the central nervous system was supported.

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[1]Eysenck (1964) in a recent article argues that reminiscence occurring after massed practice at the pursuit rotor is not due to the dissipation of reactive inhibition, but rather to the consolidation of memory traces. As an argument against the 'inhibition' hypothesis Eysenck points out that the amount of reminiscence was found to be independent of the degree of depression in pre-rest performance. Because of this discrepancy between a prediction made from the point of view of the inhibition hypothesis and experimental findings, Eysenck accepts the consolidation hypothesis, with insufficient positive evidence, by mere default of the other hypothesis. In the opinion of the author, Eysenck's findings are of great interest but his conclusion rather premature.

There were, however, decrements in pursuit rotor performance during the two massed practice periods of the post-treatment occasion for the experimental group when compared to that of the control group, while there was no difference between the two groups before treatment. The performance decrements may have been due to the confounding effects of drugs rather than to ECT but it is unlikely that drugs played a role for the following reasons. Both groups received medications at the time of the pretreatment occasion yet there were no differences in performance. Further, the drugs were fairly well randomized throughout the two groups, with the exception of antidepressants. The experimental subjects received significantly more antidepressant drugs than did the control subjects at the time of the pretreatment occasion ($\chi^2 = 7.49$; $p < .05$). There was also a greater (but nonsignificant) use of antidepressants by the experimental subjects at the time of the post-treatment occasion (see Appendix B). However, an analysis of the effect of antidepressants on pursuit rotor performance during the first occasion suggested that this type of drug facilitated performance at this task. Since the majority of subjects who used antidepressants at the time of both occasions were members of the experimental group, it is likely that the confounding effect of this type of drug would have been to increase the performance of the group after ECT, rather than to cause a performance decrement, as was the case.

It can be concluded, then, that the decrement was due to ECT. This result seems to support similar findings by Campbell (1952) who used a modified form of the Porteus mazes as a test of motor learning. He found a marked improvement in performance during the second trial before treatment, a decrement in performance during the first trial after treatment, followed by improvement on the final trial. Further, as was the case in the present study, the decrement during the post-treatment occasion was characteristic of the experimental group but not of the control group.

In reporting the results of his 1952 study, Campbell (1962) does not offer any explanation of the performance decrement. In order to understand the nature of the decrement of performance found in the present study, it is necessary to consider the interactions between the processes responsible for the increment of performance in the pursuit rotor task. The process of learning is responsible for a steady increment in performance during massed practice trials, while dissipation of reactive inhibition during a short rest after a massed practice trial produces a sharp increment in performance when the trials immediately following the rest period are compared with the trials immediately preceding the rest period.

It is the view of several investigators in the field of motor learning (e.g., Ammons, 1947; Kimble, 1952; Bilodeau, 1952) that pursuit rotor learning phenomena are best accounted for

by the inter-relationships between the four main variables in Hull's learning theory equation. Hull (1943) theorizes that two positive variables which determine the reaction potential of a learned response are the amount of drive (D) and the strength of the habit ($_{S}H_{R}$) producing the response. The reaction potential resulting from the product of these two factors is reduced by a negative drive (reactive inhibition, symbolized I_{R}) and a negative habit of not responding (conditioned inhibition, or $_{S}I_{R}$).

The relationship between these four variables which determine the reaction potential is according to Hull described by the equation $_{S}E_{R} = (D \times {}_{S}H_{R}) - (I_{R} + {}_{S}I_{R})$. This equation has been criticized by Jones (1958) who claims that the relationships between the terms of the equation are logically inconsistent because there is a multiplicative relationship between D and $_{S}H_{R}$, yet a summative relationship between a second drive (I_{R}) and a second habit ($_{S}I_{R}$). Jones proposes that the modified equation $_{S}E_{R} = (D - I_{R}) \times ({}_{S}H_{R} - {}_{S}I_{R})$ removes this logical inconsistency.

The modification by Jones has the merit that it dichotomizes the terms of the Hullian learning equation into two dimensions: that of drive and that of habit. These two dimensions of drive and habit can be related to neurophysiological functions. The drive-reactive inhibition dimension ($D - I_{R}$) corresponds to the neurological dimension of facilitation-inhibition. The condition of the central nervous system described by this dimension could then

be responsible for the magnitude of reminiscence and kinesthetic figural aftereffects, attributed previously to the accumulation of reactive inhibition or the accumulation of neural satiation, the latter of which are considered by some authorities (Eysenck, 1955; Duncan, 1956) as being the same process. The habit dimension ($_{S}H_{R}$ - $_{S}I_{R}$) correponds to the neurological mechanisms responsible for the formation of memory traces and associative learning. Pursuit rotor learning and performance can be explained by interactions between the D - I_R and $_{S}H_{R}$ - $_{S}I_{R}$ dimensions.

It can be seen from the Jones equation that a performance decrement could be due to a reduction in either drive or habit strength or to an increase in either reactive inhibition or conditioned inhibition. Referring to the performance decrement of the present study, it is unlikely that it was due to an alteration within the drive-reactive inhibition dimension because there was no change in reminiscence or figural aftereffects following ECT.[2] Hence, the reason for the decrement must be

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[2]It was predicted in this study that the central excitatory state brought about by neural facilitation would cause less reactive inhibition to be produced in massed practice learning of the pursuit rotor task and hence less reminiscence after a rest period. Some recent investigations (Eysenck and Maxwell, 1961; Eysenck and Willett, 1961; and Feldman, 1964) seem to contradict this prediction. It was found that on the pursuit rotor task more highly motivated groups, more ego-involved in the task, showed more reminiscence than did less motivated groups. The explanation offered by the above authors is that higher motivated subjects work harder at the pursuit motor task and as a result accumulate more reactive inhibition. These subjects do not "let up" during the practice periods, therefore their practice periods are more "massed" than

(Footnote continued on next page)

sought within the habit dimension. Here, the decrement could have been due to either an increase in conditioned inhibition after ECT or to a reduction in the habit strengths of the responses which had been learned prior to treatment.

According to Hull, conditioned inhibition is a learned habit not to respond in order to dissipate the accumulated reactive inhibition. Since the decrement of performance after ECT occurred on an average of 13.8 days after the previous massed practice period, and since studies have shown that most of the I_R developed during massed practice at the pursuit rotor dissipates during a ten minute rest (Kimble, 1948; and Bilodeau, 1952), it may be concluded that reactive inhibition did not play any role. Therefore conditioned inhibition, also, can be excluded.

The exclusion of conditioned inhibition leaves a decrease in habit strength as the explanation for the performance decrement. More specifically, the indications are that ECT decreases the strengths of habits associated with a previously learned task. As already mentioned in the presentation of the results, there was a significant decrement in the performance of the experimental

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(Footnote 2 continued)
those of more relaxed subjects. It is possible that in the experiments reported the central excitatory state (i.e., the position on the D - I_R continuum, to use Gwynn Jones' paradigm) of both "highly motivated" and "non-highly motivated" groups was the same and that the difference in the amount of the accumulated reactive inhibition was caused by greater massing of trials by the "highly motivated" subjects, than by the control subjects.

group when the last trials before ECT and the first trials after ECT were compared in both experimental and control groups (the $\underline{t}$ ratio for the experimental group was 4.77; $p < .001$ whereas the $\underline{t}$ for the control group was 0.91; N.S.) and also when the mean difference between trials for the two groups was compared ($\underline{t} = 1.99$; $p < .05$).

The habits were not completely forgotten since the level of initial performance during the first period of practice post-ECT was significantly higher than the level of the initial trial of the first period before treatment ($\underline{t} = 3.44$; $p < .01$). Nor was there evidence of an impairment of the capacity to learn new habits because once engaged in massed practice after ECT, the experimental subjects learned as rapidly as did the control subjects, as is indicated by the non-significant difference between the linear component of the trends of the learning curves ($F = 0.363$), and by the similarity between the groups in maximum gain in performance ($\underline{t} = 0.74$). However, this indication of unimpaired learning capacity after ECT, based as it is on a comparison of the two groups, may not be entirely representative. The performance of the control subjects was closer to asymptotic level during the second occasion than was the performance level of the experimental subjects, i.e., the experimental subjects had more room for improvement than did the control subjects, yet both groups learned at the same rate.

Since Hull's concept of habit strength likely describes the same phenomenon as does Duncan's concept of acquired habit, it

follows that the present study supports the hypothesis that ECT impairs acquired habits (Duncan, 1948) by producing a disruption of the memory traces associated with them[3]. The present study gave no support to Gellhorn's hypothesis that ECT produces a state of cortical facilitation due to stimulation of the sympathetic centers in the hypothalamus nor did it support the hypothesis that ECT produces an increased state of central inhibition held by some authors (Klein and Krech, 1952; Eysenck, 1955) to be a concomitant of brain damage.

However, the apparent untenability of these hypotheses is not conclusive. Gellhorn and Safford (1948) found that ten electroshocks were required to produce predominant sympathetico-adrenal

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[3]It is tempting to suggest that the interference with the consolidation of memory traces as postulated by Thompson and Dean (1955) is the neurophysiological process causing the disruption of acquired habits (Duncan, 1948). However, the concept of consolidation of memory trace is an ambiguous one. Some authors (Walker and Tarte, 1963) mean by this term events occurring in the nervous system during a few minutes following an exposure to a stimulus pattern. Thus these authors imply that "consolidation of memory trace" is related to the transfer of the remembered material from the immediate memory storage to the long term memory storage. Other authors (Thompson and Dean, 1955) apply the term "interference with consolidation of memory traces" to the disruption of learned habits by a brain trauma within a few hours after the learning took place. Because of this ambiguity of the term "consolidation of a memory trace" it is preferable to conclude that in the present study ECT disrupted memory traces rather than that it interfered with consolidation of memory traces.

activity in rats. The subjects of the present experiment received an average of 4.7 ECT treatments (range: 3 to 6) which may have been insufficient to produce the excitatory state. Another study which used subjects who had received ten or more treatments would be required to isolate the interactions between the number of treatments and sympathetico-adrenal activity. Further, in reference to the brain damage-inhibition hypothesis, the lack of change in reminiscence and figural aftereffects excludes the influence of ECT on the tendency to develop response-produced inhibition (i.e., reactive inhibition) but does not eliminate a possible influence of ECT on basal inhibition postulated by Klein and Krech (1952). However, although it is a theoretical possibility, according to Eysenck (1957), basal inhibition is not capable of proper experimental investigation.

That a disruption of memory traces is apparently the major effect of ECT clarifies some psychiatric clinical observations made on patients who have received the treatment. The incidence of retrograde amnesia is clearly related to the disruption of memory traces. Further, the apparent increase in 'repression' after ECT (Alexander, 1953) could be interpreted as a state of amnesia due to neurological impairment.

The disruption of memory traces can also be related to the remission of depressive illnesses. Depressive symptoms often center upon and are reinforced by ruminations on matters of personal concern

to the subject. ECT may reduce the availability of the thoughts associated with the areas of rumination so that, in effect, the subject 'cannot remember what was troubling him'. Since depressive affect is primarily a response to thought-stimuli, the reduction in the availability of such stimuli would tend to elevate the subject's mood.

CHAPTER V

SUMMARY AND CONCLUSIONS

The present study was conducted to discriminate between a cortical facilitation hypothesis and a brain damage-inhibition hypothesis of the neural effect of electro-convulsive therapy (ECT). Two measures of cortical inhibition (Ward-Hovland reminiscence and kinesthetic figural aftereffects) were used to make this discrimination. An hypothesis was advanced that a state of cortical facilitation after ECT would decrease the magnitude of reminiscence and of kinesthetic figural after-effects. It was conversely hypothesized that if ECT causes a temporary brain damage, probably producing an inhibitory state, the magnitude of reminiscence and kinesthetic figural aftereffects would increase. The findings of the present study are as follows:

1. There was no significant change in the magnitude of either reminiscence or kinesthetic figural aftereffects after ECT. Provided these two phenomena are valid measures of cortical inhibitory state, and provided the assumption that cortical inhibitory state is influenced by the level of cortical facilitation is a sound one, it may be concluded from the present study that ECT has no effect on either cortical facilitation or cortical inhibition.

2. While ECT had no effect on reminiscence or kinesthetic figural aftereffects, it did cause a decrement in pursuit rotor performance. This result supports similar findings by other

investigators. There is a strong suggestion that the performance decrement of the present study was due to an impairment of the pursuit rotor learning habit gained prior to ECT. This impairment of habit is likely due to an interference with memory mechanisms.

3. A second positive finding of the present study was a suggestion that, although ECT causes a decrement in the performance of a previously learned task, it does not appreciably alter the capacity to acquire new learning.

4. A third additional finding was a suggestion that the antidepressant drugs used by the subjects in the present study had a facilitative effect on performance at a motor learning task.

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APPENDICES

Appendix A

Sex, Age, Diagnosis and Medication of Each Subject

(a) Experimental Group

Sex and Age	Psychiatric Diagnosis	Drugs	Dosage at time of Occasion 1	Dosage at time of Occasion 2
M-39	(1) Acute Primary Depression	Elavil	25 mg TID[1]	N.A.
		Tuinal	gr.ii 10 p.m.	same
	(2) Inadequate Personality	Sodium amytal	gr.ii TID	same
F-55	(1) Neurotic Depression	Librium	N.A.	25 mg BID
	(2) Obsessive-compulsive Personality			
F-23	Acute Schizophrenic Reaction in Vulnerable Personality	Butaperazine	10 mg TID	same
		Parnate	10 mg TID	10 mg 8 p.m.
M-42	Manic-depressive, Depressive Phase	Tuinal	gr.iii 9 p.m.	same
M-55	Involutional Depression	Doriden	0.5 gm. 10 a.m.	same
		Valium	10 mg. TID	same
F-53	Neurotic Depression	Valium	5 mg. TID	same

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[1]The pharmacological abbreviations BID, TID, and QID, mean that the drug was administered twice, thrice and four times daily respectively. The abbreviations gr. i, gr. ii, gr. iii, mean grains I, II, III, respectively.

Appendix A (Cont'd.)

Sex and Age	Psychiatric Diagnosis	Drugs	Dosage at time of Occasion 1	Dosage at time of Occasion 2
F-38	Manic-depressive Disease, Depressive	Largactil	75 mg. QID	50 mg. BID
		Parnate	20 mg. TID	10 mg. TID
F-58	(1) Involutional Depression	Elavil	25 mg. 10 p.m.	same
	(2) Vulnerable Personality	Elavil	10 mg. 9 a.m.	same
F-53	Reactive Depression vs. Paranoid Personality	Elavil	10 mg. BID	same
F-51	Manic-depressive Reaction, Depressive Type	Largactil	100 mg. 10 p.m.	50 mg. 10 p.m.
		Nardil	30 mg. TID	N.A.
		Stelazine	2 mg 8 a.m., 5 p.m.	N.A.
F-44	Depressive Reaction	Largactil	100 mg. 10 p.m.	same
		Parstellin	1 tab. BID	same
F-58	Endogenous Depression, with Depressive Elements	Phenobarbital	gr. 1/4 QID	same
		Nardil	1 tab. BID	N.A.
F-42	Paranoid Personality, Psychotic Reaction	Tofranil	50 mg. TID	same
		Largactil	75 mg. QID	same
		Stelazine	5 mg. TID	2 mg. TID
F-29	(1) Depressive Reaction	Parnate	10 mg. 8, 10, 12 a.m.	same
	(2) Anxiety Reaction	Largactil	75 mg. QID	same

Sex and Age	Psychiatric Diagnosis	Drugs	Dosage at time of Occasion 1	Dosage at time of Occasion 2
M-39	Acute Psychotic Depression	Librium	10 mg. TID	same
		Parnate	N.A.	10 mg. 9 a.m.
(b) Control Group				
F-33	Depression	Stelazine	5 mg. BID	N.A.
		Equanil	N.A.	200 mg. TID
F-30	Neurotic Anxiety Reaction	Librium	10 mg. TID	same
F-21	Hypomania	Sparine	50 mg. TID	same
F-47	Character Disorder, Depressive Reaction	Stelazine	5 mg. TID	same
F-32	Schizoid Personality	Tofranil	25 mg. TID	50 mg. TID
		Largactil	25 mg. TID	N.A.
		Mellaril	N.A.	25 mg. TID
M-26	Obsessive-compulsive Personality with Schizophrenic Episodes	Stelazine	0.5 mg. TID	same
F-31	Acute Schizophrenic Reaction	Stelazine	N.A.	2 mg. BID
		Tofranil	N.A.	25 mg. TID
M-27	Latent Homosexuality	N.A.		
F-24	Paranoid Schizophrenia	Stelazine	5 mg. TID	N.A.

Sex and Age	Psychiatric Diagnosis	Drugs	Dosage at time of Occasion 1	Dosage at time of Occasion 2
F-23	Reactive Depression	Stelazine	5 mg. TID	same
F-23	(1) Dependent Personality	Valium	5 mg. TID	same
	(2) Reactive Depression			
F-49	Involutional Depressive Reaction	Stelazine	N.A.	2 mg. 1 x daily
F-49	Schizo-affective Psychosis	N.A.		
F-39	Severe Sado-masochistic Marital inter-action with Depressive Reaction, with some Paranoid Features, to her Personality	Valium	5 mg. TID	same
		Librium	N.A.	10 mg. TID
F-43	Neurotic Depressive Reaction	Librium	10 mg. TID	same
		Tofranil	25 mg. TID	same

Distributions of Subjects in Each Group Who Received Tranquilizer, Antidepressant or Sedative Drugs

(1) Tranquilizers

Group	Dosage at Time of Occasion 1				Dosage at Time of Occasion 2		
	0	1	2		0	1	2
ECT	6	4	5		6	6	3
Control	4	10	1		4	10	1

(2) Antidepressants

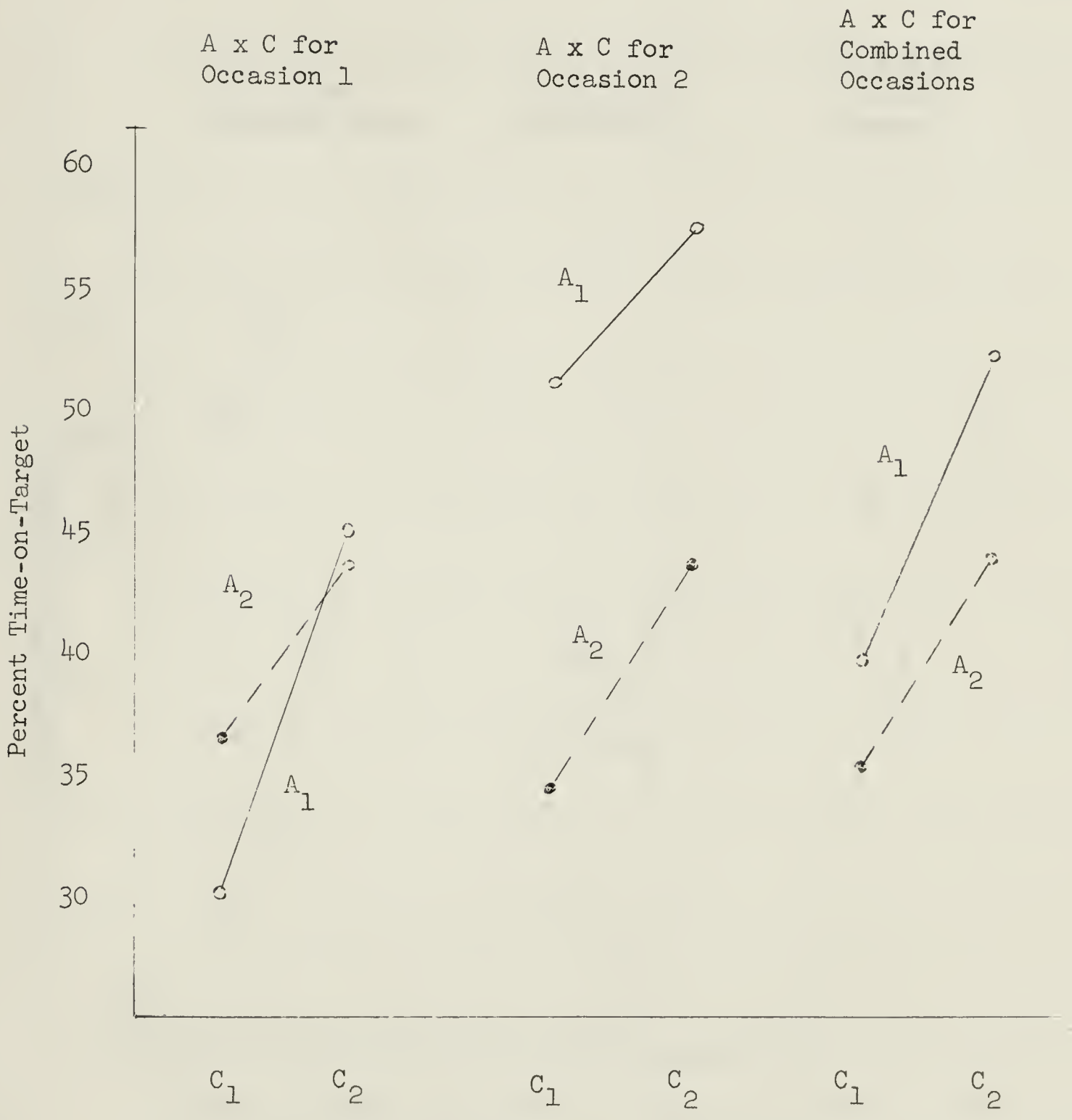
Group	Dosage at Time of Occasion 1				Dosage at Time of Occasion 2		
	0	1	2		0	1	2
ECT	6	6	3		7	6	2
Control	13	2	0		12	2	1

(3) Sedatives

Group	Dosage at Time of Occasion 1				Dosage at Time of Occasion 2		
	0	1	2		0	1	2
ECT	11	4	0		12	3	0
Control	15	0	0		15	0	0

Appendix C

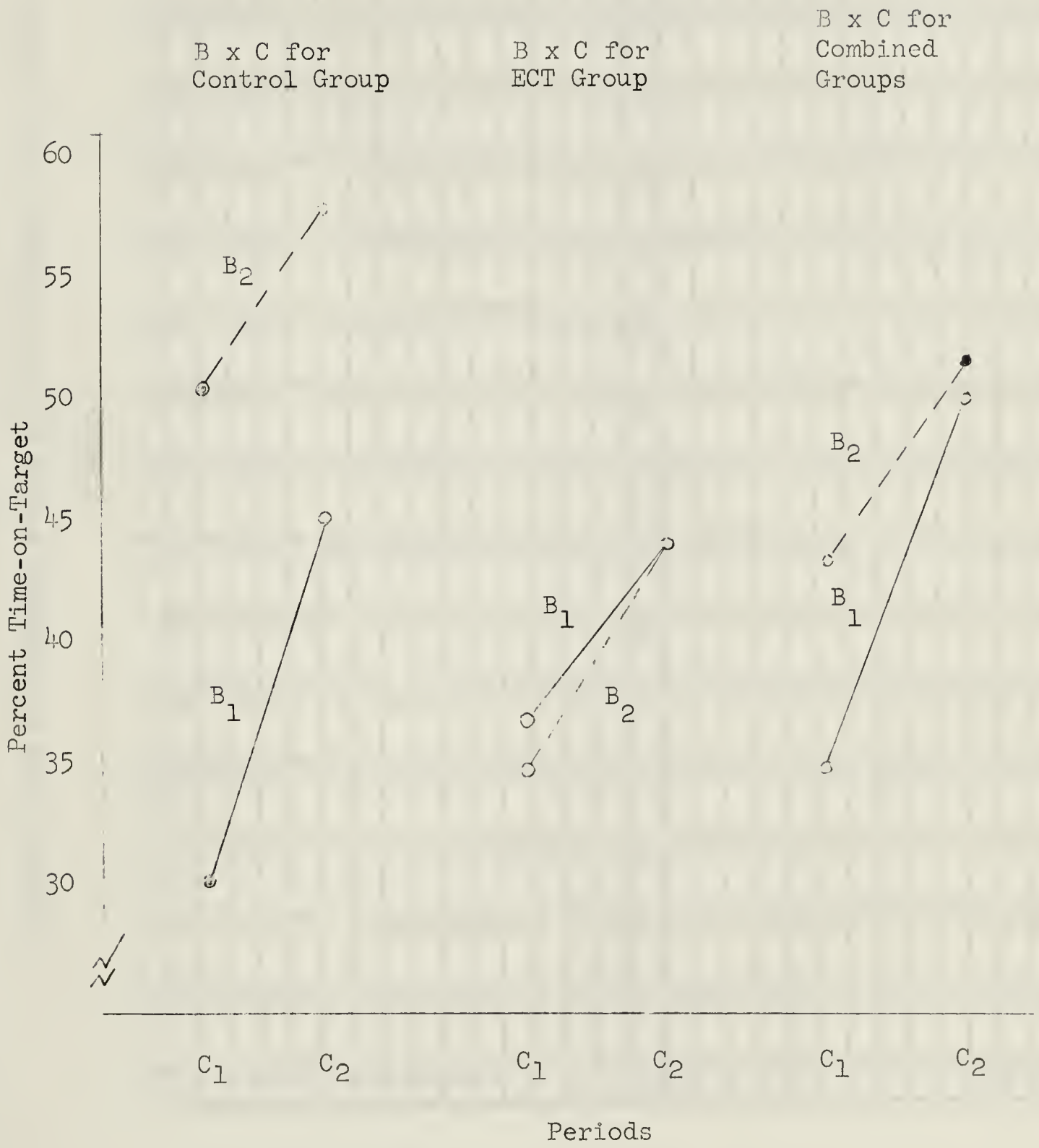
(a) Groups by Periods Interactions of Reminiscence Scores



A_1 = Control Group

A_2 = Experimental Group

(b) Occasions by Periods Interactions of Reminiscence Scores



B_1 = Occasion 1
B_2 = Occasion 2

Appendix D

Pursuit Rotor Performance Scores for Both Groups During Period 1

	TRIALS															
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
A_1 (Control)	8	5	12	5	16	27	21	26	17	24	24	18	23	21	16	263
	1	0	0	1	0	3	1	2	6	10	7	4	26	18	20	99
	2	3	5	12	13	18	21	19	22	31	34	31	34	21	22	288
	12	11	7	17	16	14	19	26	23	30	36	25	31	38	36	341
	0	2	1	1	2	9	12	7	5	5	9	7	4	3	4	74
	1	1	1	1	2	10	8	20	20	17	21	12	7	11	11	143
	4	2	7	7	7	8	4	9	9	25	21	15	17	30	15	180
	15	10	20	19	21	17	23	18	20	32	44	38	19	16	16	328
	12	6	37	44	32	37	39	27	49	45	45	29	52	46	49	549
	29	53	66	78	45	60	77	70	68	84	72	77	78	79	64	1030
	15	12	26	15	15	14	12	15	14	13	13	22	28	22	26	262
	6	7	17	28	35	26	24	20	34	38	35	44	53	52	50	469
	4	3	9	22	26	38	50	45	11	25	47	29	37	17	27	390
	14	16	22	22	26	29	25	31	32	34	33	30	45	34	42	435
	7	42	46	51	58	46	47	56	47	62	51	61	56	52	58	740
ΣA_1	130	173	276	323	344	356	383	391	377	475	492	442	510	460	456	5588
A_2 (ECT)	22	34	46	43	40	16	10	17	22	34	38	38	47	44	54	505
	7	20	24	22	20	31	17	22	53	20	22	34	20	22	25	359
	4	8	14	18	40	26	20	32	46	35	33	20	27	30	45	398
	25	38	40	54	53	27	44	33	34	50	45	42	36	41	31	593
	7	16	20	24	33	35	29	44	43	36	43	40	28	53	57	508
	4	2	11	20	24	20	23	34	29	35	41	40	39	26	32	380
	1	5	4	7	4	10	6	4	5	7	7	10	28	13	10	121
	9	22	19	31	19	22	29	14	37	25	14	15	19	20	24	319
	2	2	2	6	13	24	13	31	27	28	14	16	11	26	20	235
	0	1	3	4	5	16	6	7	12	10	4	5	8	6	2	89
	19	27	39	45	55	50	37	50	46	32	42	48	46	51	40	627
	40	45	53	64	54	58	64	57	60	49	41	34	41	39	32	731
	44	67	63	69	75	74	66	65	74	73	66	75	70	70	74	1025
	3	18	22	32	39	40	34	42	45	30	34	29	19	28	32	447
	6	14	29	23	34	44	31	41	32	53	30	31	70	68	70	577
$\bar{X}_{A_1}$	8.7	11.5	18.4	21.5	22.9	23.7	25.5	26.1	26.4	31.7	32.8	29.9	39.2	30.7	30.0	
$\bar{X}_{A_2}$	12.8	21.3	25.9	30.8	33.9	32.9	28.5	32.9	37.7	34.5	31.2	31.8	33.9	35.8	36.5	
ΣA_2	193	319	389	462	508	493	429	493	565	517	474	477	509	537	548	6914
Σ_R	323	492	665	785	852	849	812	884	942	992	966	919	1019	997	1004	12502

Pursuit Rotor Performance Scores for Both Groups During Period 2

	TRIALS															
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
A_1 (Control)	36	46	55	40	47	42	44	39	49	55	47	33	39	78	53	703
	25	34	36	36	35	20	42	23	26	27	23	18	21	15	16	398
	48	52	62	62	67	61	72	49	52	48	63	45	65	53	58	857
	45	43	55	62	55	65	65	56	62	50	60	54	63	62	57	854
	15	22	28	16	21	28	26	19	27	24	22	7	20	37	18	330
	19	29	30	25	26	21	18	19	24	32	13	24	33	27	24	364
	45	52	56	54	57	59	61	54	55	52	57	55	50	56	48	811
	39	47	27	21	56	32	48	51	52	63	59	67	57	49	59	727
	64	78	72	75	56	68	68	83	65	76	73	51	66	69	62	1026
	74	84	85	91	82	100	89	93	91	89	84	90	93	87	79	1311
	60	47	58	57	45	43	36	57	58	51	47	45	46	47	41	738
	49	63	69	68	63	56	61	60	46	38	53	50	53	63	43	835
	59	56	76	77	76	71	56	63	46	61	67	43	52	46	64	913
	55	63	61	63	69	65	67	77	63	53	69	64	69	68	56	962
	46	60	78	64	78	77	79	78	77	79	80	71	70	41	64	1042
$\bar{X}_{A_1}$	45.26	51.8	56.6	54.4	55.5	53.9	55.5	54.7	52.9	53.2	54.5	47.8	53.1	53.2	48.5	
ΣA_1	679	776	848	811	833	808	832	821	793	798	817	717	797	798	742	11870
A_2 (ECT)	60	65	72	65	55	62	56	73	68	57	55	56	53	49	54	900
	46	54	54	64	70	73	66	58	65	61	61	69	68	61	65	935
	28	40	18	27	43	31	38	22	17	21	11	35	26	29	21	406
	59	69	59	71	72	77	58	64	68	71	57	64	71	68	67	995
	63	47	59	57	36	45	60	50	62	49	47	65	70	68	41	819
	55	61	68	55	62	77	74	65	71	67	69	69	64	55	59	971
	68	71	53	55	72	79	71	67	75	77	76	78	64	84	79	1069
	30	32	38	35	40	36	46	61	38	28	43	35	40	43	34	579
	64	78	83	84	84	86	87	89	91	88	80	86	85	85	80	1250
	50	30	43	56	59	60	64	72	40	60	32	69	58	49	47	789
	48	55	55	55	57	62	62	60	53	61	61	59	52	57	59	856
	13	20	12	15	14	11	13	15	16	20	25	13	12	14	20	233
	25	20	27	14	24	21	27	33	34	25	35	23	24	30	27	389
	24	24	23	29	32	35	21	23	21	23	29	15	18	36	20	373
	27	32	27	36	32	32	39	37	39	28	25	38	38	48	41	519
ΣA_2	660	698	691	719	752	787	780	789	758	736	706	774	743	776	714	11083
$\bar{X}_{A_2}$	43.9	46.5	46.0	47.9	50.1	52.5	52.0	52.6	50.5	49.7	47.1	51.7	49.5	52.2	47.6	
ΣB	1339	1474	1539	1530	1585	1595	1612	1610	1551	1534	1523	1491	1540	1574	1456	22953

Appendix D (Con'd.)

Pursuit Rotor Performance Scores for Both Groups During Period 3

	TRIALS															
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
A_1 (Control)	44	41	46	44	49	46	42	50	48	43	36	30	50	46	43	658
	28	37	29	23	24	20	25	25	31	23	26	30	27	42	47	437
	18	16	6	11	7	6	27	22	18	18	19	30	12	17	15	242
	46	59	53	58	79	77	60	75	59	64	65	67	81	68	68	979
	40	47	62	36	32	49	37	34	40	21	31	34	25	22	29	539
	21	39	38	47	46	40	43	36	31	31	22	36	29	28	37	524
	42	46	48	50	66	70	53	57	53	66	58	52	56	68	55	840
	31	30	46	53	53	64	60	55	62	60	52	44	44	52	52	758
	18	29	50	49	52	33	47	42	19	41	47	45	53	41	23	589
	72	78	83	80	74	87	79	78	80	80	73	74	73	83	80	1167
	58	57	68	67	68	73	70	71	69	71	60	69	69	69	66	1005
	81	79	81	82	73	46	78	79	62	75	62	71	73	73	53	1068
	58	61	69	72	76	68	70	61	67	78	57	55	67	67	70	996
	58	56	64	51	60	58	45	50	55	48	47	46	54	63	57	812
	51	80	72	70	80	80	77	71	80	75	81	68	79	78	66	1108
$\bar{X}_{A_1}$	44.0	50.3	54.3	52.8	55.9	54.5	54.2	53.3	51.6	53.0	49.1	50.0	50.8	54.5	50.7	
ΣA_1	666	755	815	793	839	817	813	799	774	794	736	751	792	817	761	11722
A_2 (ECT)	28	28	44	41	44	52	45	55	43	55	48	53	52	42	47	677
	30	40	36	36	46	43	41	35	47	51	45	46	33	29	5	563
	31	37	43	42	48	36	37	40	28	19	21	21	15	17	43	478
	17	11	21	20	29	33	46	35	45	52	38	45	41	42	37	512
	29	28	10	27	29	48	34	26	24	17	18	15	17	13	21	356
	44	29	17	27	40	36	44	41	46	46	49	34	39	46	28	566
	21	18	21	28	26	29	29	19	28	22	32	25	24	30	32	384
	3	3	1	0	0	1	0	1	1	2	2	2	1	6	2	25
	24	26	19	29	32	30	38	34	41	32	39	42	39	33	31	489
	4	7	8	10	15	11	4	14	13	18	20	8	10	21	22	185
	61	56	58	69	71	63	68	62	54	70	70	53	78	61	65	959
	7	39	41	39	38	43	48	58	48	24	38	28	29	28	34	542
	34	72	75	80	78	80	75	66	81	80	75	77	81	74	78	1106
	47	57	64	55	71	72	62	14	75	73	70	68	75	62	51	916
	43	48	61	61	68	50	67	52	44	45	41	51	48	48	21	748
ΣA_2	423	499	519	564	635	627	638	552	618	605	606	568	582	552	518	8506
$\bar{X}_{A_2}$	28.1	32.6	33.5	37.6	42.3	41.8	42.5	36.8	41.2	40.4	40.4	37.9	38.8	36.8	34.4	
ΣB	1089	1254	1334	1357	1474	1444	1451	1351	1392	1399	1342	1319	1374	1369	1279	20228

Appendix D (Cont'd.)

Pursuit Rotor Performance Scores for Both Groups During Period 4

	TRIALS															
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
A_1 (Control)	58	55	53	56	60	62	65	65	62	64	61	59	76	67	62	925
	54	54	57	52	51	49	55	66	59	51	58	30	37	47	42	762
	39	46	46	62	48	57	47	53	60	41	52	57	63	50	52	773
	65	74	84	80	74	78	82	81	73	69	80	75	77	76	69	1137
	49	59	50	54	45	53	51	45	40	41	47	45	58	37	40	714
	41	55	49	52	58	53	48	45	48	43	39	51	42	48	48	718
	55	65	70	71	61	70	64	69	76	73	72	70	70	72	67	1025
	38	52	54	52	46	45	52	43	43	50	45	52	43	42	39	696
	49	54	60	62	67	67	69	51	67	42	49	60	52	49	53	851
	72	85	82	87	91	89	88	90	84	91	95	84	90	85	84	1297
	68	83	87	86	91	86	86	91	89	85	92	88	82	80	84	1278
	83	77	87	87	89	89	81	89	88	83	81	72	84	86	87	1263
	67	70	75	72	82	73	83	76	69	76	72	75	71	66	69	1096
	61	79	71	60	59	63	78	65	58	73	78	67	55	46	58	971
	78	79	89	84	80	81	86	84	85	79	83	83	83	73	89	1236
$\bar{X}_{A_1}$	58.5	65.8	67.6	67.8	66.8	67.0	69.1	67.6	66.6	64.1	66.9	64.5	65.5	66.6	62.3	
ΣA_1	877	987	1014	1017	1002	1015	1035	1013	999	961	1004	968	983	924	943	14742
A_2 (ECT)	39	37	44	62	63	56	61	40	54	69	55	62	66	57	55	820
	28	29	35	33	37	40	41	28	22	46	38	18	43	31	39	508
	52	67	59	74	56	63	53	60	66	50	61	63	40	48	50	862
	49	51	62	61	57	60	64	57	71	71	63	62	47	40	43	858
	41	38	51	45	51	50	65	48	45	44	58	53	33	42	41	705
	62	60	54	56	70	45	52	63	59	36	58	45	53	46	37	796
	48	37	39	27	55	44	55	89	72	38	57	55	56	42	58	732
	72	67	66	80	76	74	67	69	74	74	64	71	71	62	65	1052
	74	78	78	82	82	83	81	81	81	80	80	75	78	79	78	1190
	28	54	50	50	43	39	38	49	51	23	50	48	70	29	26	648
	56	58	66	74	62	73	68	75	67	71	63	65	70	73	71	1012
	31	22	26	33	35	19	16	23	20	22	23	19	17	18	20	344
	39	41	55	57	47	51	47	38	50	41	46	44	45	43	34	678
	3	1	0	3	5	1	6	4	6	14	5	20	16	28	20	132
	38	27	25	39	33	30	33	29	27	33	31	38	40	47	38	508
ΣA_2	660	667	710	776	772	728	747	713	765	712	752	738	745	685	675	10845
$\bar{X}_{A_2}$	44.0	44.5	47.3	51.7	51.5	48.5	49.8	47.5	51.0	47.5	50.0	49.2	49.6	45.7	45.0	
ΣB	1537	1654	1724	1793	1774	1743	1782	1726	1764	1673	1756	1706	1728	1609	1618	25587

Appendix E

Kinesthetic Figural Aftereffect Data for Control Group

Wedge Position and Direction Approach	Subjects	Occasion 1			Occasion 2			KAE Difference
		pse_1	pse_2	KAE	pse_1	pse_2	KAE	
B_n[1]	1	1 14/16	1 12/16	+ 2/16	1 14/16	1 14/16	0	+ 2/16
	2	1 4/16	1 7/16	- 3/16	1 6/16	1 6/16	0	- 3/16
	3	1 10/16	1 16/16	- 6/16	2 1/16	1 13/16	+ 4/16	- 10/16
	4	1 13/16	1 10/16	+ 3/16	2 2/16	1 10/16	+ 8/16	- 5/16
	5	2 6/16	1 16/16	+ 6/16	1 16/16	2 2/16	- 2/16	+ 8/16
F_n	6	2 8/16	2 1/16	+ 7/16	2 1/16	2 2/16	- 1/16	+ 8/16
	7	2 2/16	1 16/16	+ 2/16	2 3/16	1 15/16	+ 4/16	- 2/16
B_w	8	1 15/16	1 15/16	0	1 10/16	1 13/16	- 3/16	+ 3/16
	9	2 2/16	2 3/16	- 1/16	2 2/16	1 16/16	+ 2/16	- 3/16
	10	2 2/16	1 12/16	+ 6/16	2 4/16	2 4/16	0	+ 6/16
	11	1 16/16	1 16/16	0	2 2/16	1 14/16	+ 4/16	- 4/16
F_w	12	2 2/16	2 4/16	- 2/16	2 4/16	1 14/16	+ 6/16	- 8/16
	13	1 14/16	1 14/16	0	1 14/16	1 12/16	+ 2/16	- 2/16
	14	1 15/16	1 8/16	+ 7/16	1 14/16	1 11/16	+ 3/16	+ 4/16
	15	2 8/16	1 16/16	+ 8/16	2 6/16	1 15/16	+ 7/16	+ 1/16

[1]The procedural conditions represented by the symbols are as follows:
B_n = Wedge in backward position, approach from the narrow end
F_n = Wedge in forward position, approach from the narrow end
B_w = Wedge in backward position, approach from the wide end
F_w = Wedge in forward position, approach from the wide end

Appendix E (Cont'd.)

Kinesthetic Figural Aftereffect Data for Experimental Group

Wedge Position and Direction Approach	Subjects	Occasion 1			Occasion 2			KAE Difference
		pse_1	pse_2	KAE	pse_1	pse_2	KAE	
B_n [1]	1	1 11/16	1 11/16	0	1 12/16	1 10/16	+ 2/16	- 2/16
	2	1 16/16	1 13/16	+ 3/16	1 16/16	1 16/16	0	+ 3/16
	3	1 16/16	1 12/16	+ 4/16	1 16/16	1 16/16	0	+ 4/16
F_n	4	1 16/16	1 14/16	+ 2/16	1 16/16	1 10/16	+ 4/16	- 2/16
	5	1 14/16	1 13/16	+ 1/16	1 13/16	1 6/16	+ 7/16	- 6/16
	6	1 14/16	1 10/16	+ 4/16	1 15/16	1 9/16	+ 6/16	- 2/16
	7	2 2/16	1 11/16	+ 3/16	1 16/16	1 12/16	+ 4/16	- 1/16
B_w	8	2 6/16	1 14/16	+ 8/16	1 15/16	1 10/16	+ 5/16	+ 3/16
	9	2 8/16	2 8/16	0	1 14/16	1 12/16	+ 2/16	- 2/16
	10	2 6/16	2 4/16	+ 2/16	2 3/16	1 16/16	+ 3/16	- 1/16
F_w	11	2 4/16	2 4/16	0	2 6/16	2 6/16	0	0
	12	2 3/16	2 2/16	+ 1/16	2 4/16	2 6/16	- 2/16	+ 3/16
	13	2 3/16	1 11/16	+ 4/16	1 14/16	1 10/16	+ 4/16	0
	14	1 16/16	1 11/16	+ 5/16	1 14/16	1 12/16	+ 2/16	+ 3/16
	15	1 14/16	1 16/16	- 2/16	1 16/16	1 13/16	+ 3/16	- 5/16

[1] The procedural conditions represented by the symbols are the same as those described in the footnote of the previous table.

Appendix F

The ECT equipment consisted of an Ectron electro-convulsive apparatus (Serial No. 269) which delivered a direct current of 90 volts for approximately one second (the length of the impulse was manually determined). The grand mal convulsion was modified by intravenous administration of atropine (1/100 gr.); pentothol (2.5% or 2 mg./cc; 5-10 cc. per patient) and anectine 0-40

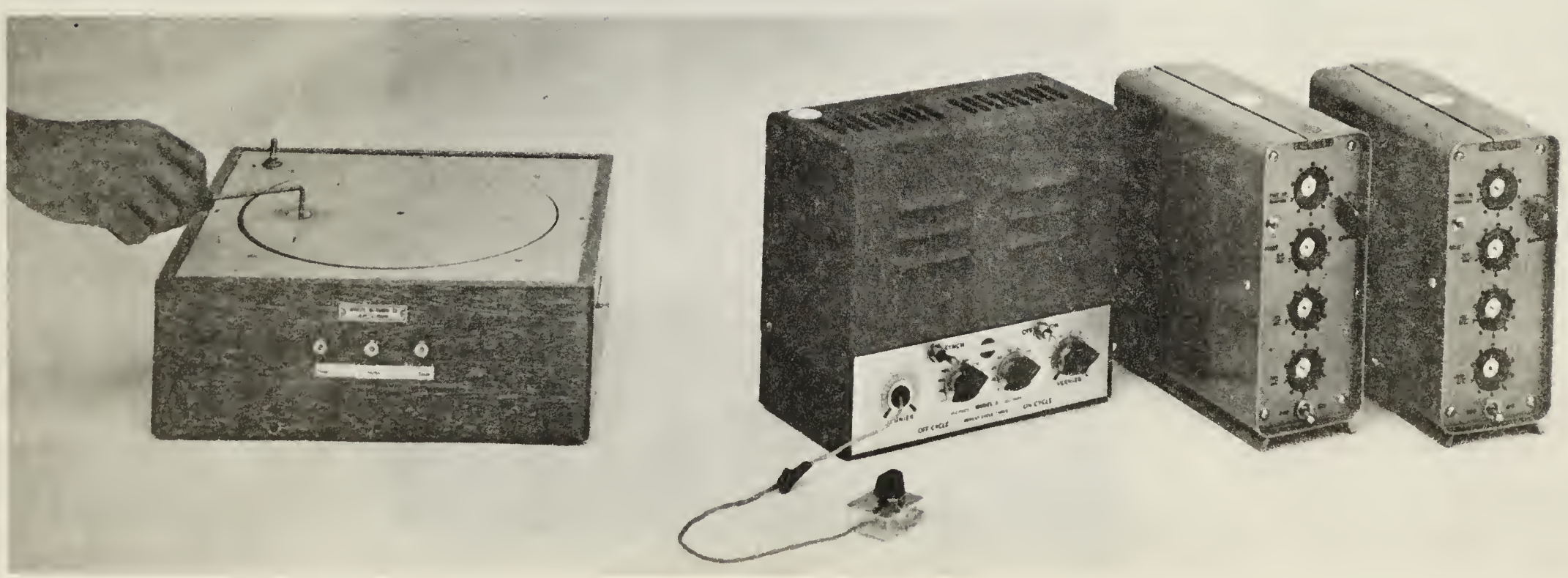
Appendix G

Pursuit Rotor Apparatus

Pursuit Rotor

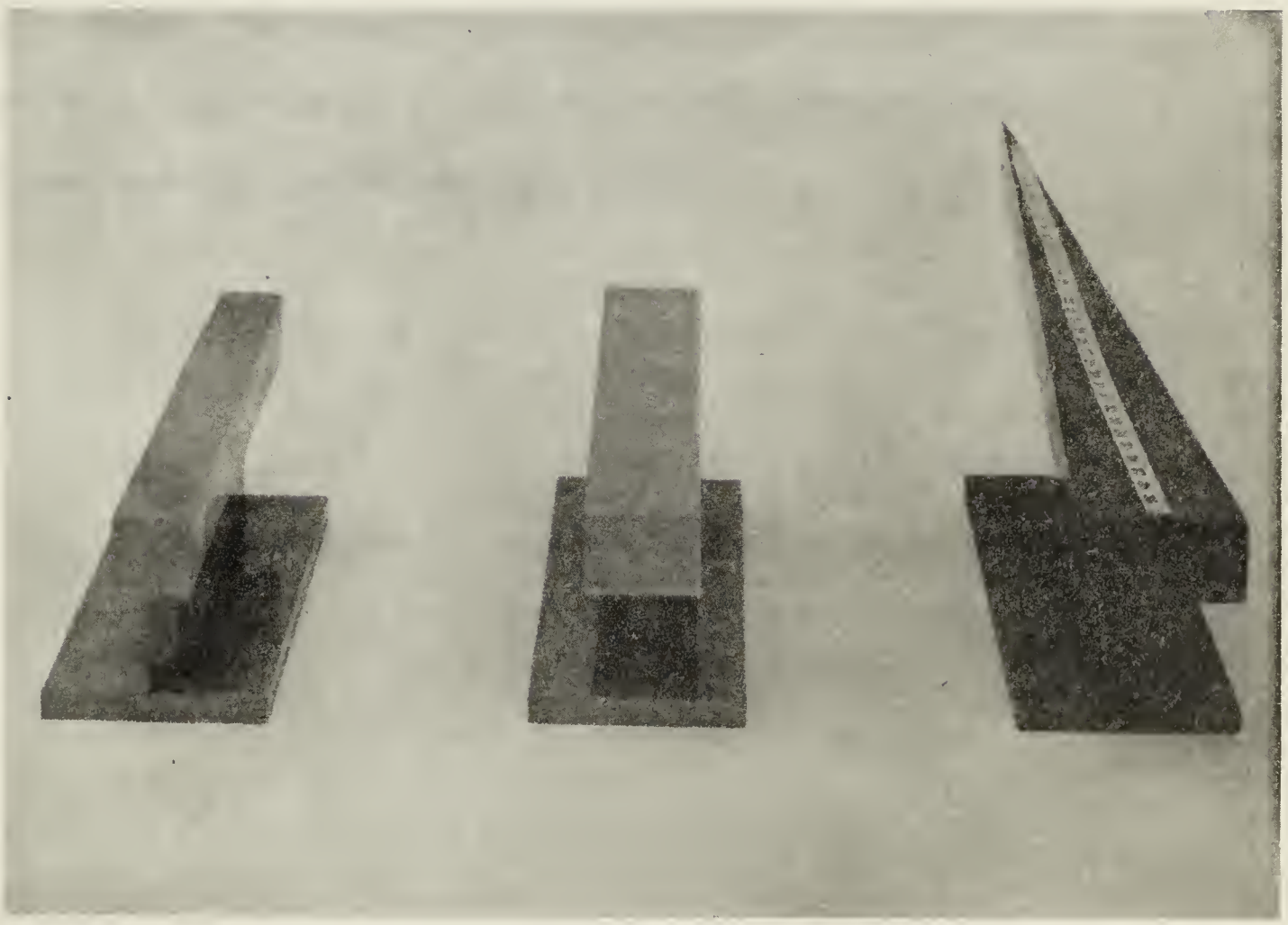
Repeat Cycle Timer

Hunter Timers



Kinesthetic Figural Aftereffect Equipment

Test Block Inspection Block Wedge



Appendix H

Effect of Antidepressant Drugs on Reminiscence

During the thesis oral examination an inquiry was made regarding the possible effect of antidepressant drugs on reminiscence. A subsequent analysis of the data showed that the mean reminiscence score for the antidepressant groups (N = 11) was 9.27 whereas the average reminiscence score for the control group (N = 19) was 12.47. The difference between these two scores was not significant (t = 0.602).